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1907.

QUEENSLAND.

DEPARTMENT OF PUBLIC HEALTH.

REPORT

ON

PLAGUE IN QUEENSLAND,

1900-1907

(26th FEBRUARY, 1900, to 30th JUNE, 1907),

BY

B. BURNETT HAM,
M.D., M.R.C.S., D.P.H. (CAMB.),
FELLOW OF THE ROYAL SANITARY INSTITUTE,
COMMISSIONER OF PUBLIC HEALTH.

PRESENTED TO BOTH HOUSES OF PARLIAMENT BY COMMAND.

BRISBANE:
BY AUTHORITY: GEORGE ARTHUR VAUGHAN, GOVERNMENT PRINTER, WILLIAM STREET.
1907.

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for Queensland.

Department of Public Health,
Brisbane.

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Office of the Commissioner of Public Health,
Brisbane, 2nd September, 1907.

TO THE UNDER SECRETARY, HOME SECRETARY'S DEPARTMENT.

SIR,—I have the honour to submit a Report on Plague in the State of Queensland for the period from 26th February, 1900, to 30th June, 1907, inclusive.

This Report narrates the accumulated experience of eight successive years, and describes at length the origin and nature of the disease, its mode of spread, and the whole of the circumstances which accompanied its appearance. Its issue has been postponed partly from causes which were unavoidable, and partly for the purpose of affording time for the completion of certain experimental work instituted with the view of establishing our knowledge of Plague on a firmer basis of fact than would, without such work, have been possible; and, while the magnitude of the work has increased beyond the limits originally intended, such increase is largely due to the fact that the scientific treatment of the subject has been rendered possible by quite recent discoveries. In fact, it is only within the past seven or eight years, *i.e.*, since the advent of Plague in Australia—that many facts essential to the knowledge of the disease have been recorded. The several reports of the Chief Medical Officer of the Government of New South Wales, Dr. J. Ashburton Thompson, on the outbreaks of Plague in that State during 1900-1905, occupy over 200 folios, and constitute a most valuable contribution to the epidemiological literature on the subject.

My object has been to embody in one continuous Report the results of our own special inquiries into the origin of the many but, fortunately, limited outbreaks in various important centres of the State, and to hand down to my successors in office as complete a history of Plague, observed in Queensland, as it has been possible to produce, so that there may be a full record of experience behind future action in order to prevent the mistakes and errors unavoidable in its absence.

We are not now so comparatively ignorant of the subject as we were when Plague first made its appearance in our midst, at the beginning of the year 1900. Many able authorities have, in the meanwhile, made considerable research into the disease, but there is still required a more complete scientific investigation of Plague apart from mere laboratory inquiries. Thus it is of the greatest importance in this connection that, accepting the rat-flea hypothesis of Plague dealt with at length in this Report, the habits and distribution of the rat, and the life-history of the rat-flea, be minutely investigated.

Plague has already been the subject of two International Conferences, and a third Conference has lately been convened. Professor Kitasato, of Japan, who in 1894 discovered the bacillus of Plague as the outcome of his investigations at Hong Kong, has stated that the expenditure of "efforts, however laborious, the money however vast, can be of no value unless they are accompanied by the application of scientific knowledge."

To return to the Report itself. It has been divided into four parts.

Part I. sets forth the manner in which Plague was introduced into Queensland, together with a general review of the various epidemies which occurred throughout the State from the beginning of 1900 to 30th June, 1907. In this portion of the Report will be found also much statistical data.

Part II. deals with the Incidence of the Plague on sex, age, race, occupation, and as to how far seasonal and insanitary conditions affect the liability of the individual to Plague infection.

In Part III. will be found a detailed description of the medical aspects of Plague, the clinical features and clinical forms of the disease, the bacteriology and pathology, and the treatment and prophylaxis of Plague; these subjects being discussed at some length.

Part IV. is confined to the Epidemiology of Plague, and a consideration of the many problems which arise in connection therewith. This portion of the Report is divided into two sections: Section one dealing with Rats and Rat-plague, and section two with the Flea Theory and the Parasites on Rats (Fleas). The latter section has been written in conjunction with Mr. Henry Tryon, the Government Entomologist, who is chiefly responsible for the particular subjects dealt with under a series of separate headings, and to whom I desire to express my sincere thanks for the valuable information so readily and ably supplied by him. To Mr. W. H. A. Munro, of the Department, I am also specially indebted. His excellent work in the preparation of the numerous maps, plans, charts, and diagrams contained in the Report reflects the greatest credit upon him. To Mr Munro also fell much arduous work in compiling the statistical data and tabular statements.

The Government Bacteriologist (Mr. C. J. Pound) carried out, from time to time, much valuable research work, and a description of the observations made by him will be found under various headings of and Appendices to the Report.

I also beg to express my thanks to Dr. Frank Tidswell, the Principal Assistant Medical Officer of the Government of New South Wales, for advice with regard to portions of the Report, and also to Drs. A. Jeffries Turner and Wilton Love for their kindness in revising the proof sheets of Parts I., II., and III. To Dr. Thomas Bancroft for the microphotographs of fleas taken from Brisbane rats, to all the medical officers who have been connected with the Department during the period under review, and to the Inspectorial and Sanitary Staffs, I tender my sincere thanks for the generous co-operation and assistance given to me.

I have the honour to be,

Sir,

Your obedient Servant,

B. BURNETT HAM,

Commissioner of Public Health.

PART I.

A.—THE STATISTICS OF PLAGUE.

I.—ORIGIN AND MODE OF SPREAD.

On 19th January, 1900, plague was reported to have broken out at Sydney, which city lies some 500 miles south of Brisbane, and distant only one and a-half days by steamer. It was subsequently ascertained by the health authorities at Sydney that rats had been dying in great numbers upon one of the wharves about the first week in January, 1900. On 14th February, seven plague-infected rats were obtained from one Sydney wharf. It seems clear, then, that an epizoötic among rats commenced at Sydney at a date prior to the occurrence of the first case in a human being.

The origin of the outbreak in Queensland is ascribed to the importation of infection, conveyed through the agency of rats, from vessels arriving at Queensland ports from Sydney at a time when plague was known to exist at the latter place.

In view of the close and constant commercial communication between Sydney and Brisbane, the health authorities and members of the medical profession, at Brisbane, were on the alert, and it was resolved at a special meeting of the Queensland Central Board of Health, on the 26th February, 1900, that the local authorities of the Metropolitan Area should combine together in a crusade to exterminate the rats of Brisbane; and, further, that all rats found sick or dead be submitted to bacteriological examination. On 5th March, 1900, the Government Bacteriologist (Mr. C. J. Pound) made the unwelcome discovery that a black rat (*Mus rattus*), found dead in a shop close to a wharf at which vessels from Sydney had recently arrived, was infected with plague. It was not, however, till the 27th April, 1900, that a plague case occurred among human beings at Brisbane. This case was not the first case of plague in man in Queensland. On 17th April, 1900, a man, the head steward on board the ss. "Burwah," a coastal vessel carrying passengers and cargo between Sydney and Rockhampton, and calling *en route* at Brisbane, was reported by Dr. Davidson, of Rockhampton, the acting Health Officer, to be suffering with symptoms suggestive of plague. The ss. "Burwah" had arrived at Rockhampton early the day before, the 16th April, having left Brisbane on the 13th April and Sydney on the 10th April.

From the official report, it appears that the steward was taken suddenly ill on board about 6 o'clock on the evening of 15th April, but his illness was not reported to the captain of the vessel until breakfast time the next morning. Dr. Davidson, who examined the patient in the absence of Dr. Voss, the Health Officer to the port, "found the patient with a temperature of 105 degrees Fahr. and a mass of swollen glands in the left groin." The ship was ordered into quarantine at McKenzie Island, Keppel Bay. Dr. D. McDonald, Medical Officer of Health to the Municipality of Rockhampton, who also saw the case in consultation with Dr. Davidson, agreed with the latter's opinion that the case was one suspicious of plague. Smear preparations from material taken from the affected glands were forwarded to Brisbane and examined by the Government Bacteriologist with positive results. The patient was subsequently examined by Dr. A. J. Turner, the special medical officer appointed by the Government for plague, at the Quarantine Hospital, Rockhampton, on 6th May, 1900.

Dr. Turner reported:—"Meyer, steward, ss. 'Burwah,' states that his illness began suddenly on 15th April with vomiting and giddiness. On the 16th instant his general condition was much the same, and he noticed for the first time a tender lump in the left groin. That day he came under the observation of the port health officer. From notes taken, I find that the highest temperature recorded was 105 degrees Fahr., the lowest 101 degrees Fahr., and he was slightly delirious. On the 17th instant his temperature was continuously 105 degrees, except when reduced by wet packs. On the 19th his temperature was 105 degrees, and he was very delirious; on the 20th the temperature was 102·8 degrees, and on 21st it was normal, and has remained so since.

"Present condition (6th May).—There is a softening mass formed by the left femoral gland, which is not now tender. A puncture was made in the affected gland and smear preparations made. These were subsequently stained and found to contain plague bacilli."

As this patient was taken ill a day prior to the arrival of the vessel at Rockhampton, it is fair to assume that he contracted the disease either at Brisbane—where rats had been found dead of plague on the wharves since 5th March, but where no case had as yet occurred in human beings—or at Sydney, where an epizoötic and an epidemic were both present at the time the vessel left on the 10th of April, 1900. It may be pointed out, however, that communication with Sydney had been steadily maintained by the coastal trade for months subsequent to the discovery of plague-infected rats in Sydney in the beginning of that year, and that the appearance of the disease among the rats on board the vessel was likely to be attended with risk to both passengers and crew, and with the likelihood of importation of the infection to hitherto clean ports in Queensland. In the official report of Dr. McDonald, it is stated: "On removal of the lining of the cabin occupied by the patient on the ss. 'Burwah,' several dead rats were found." Information concerning the mortality among rats at Rockhampton was furnished by Dr. Turner in a report to the Honourable the Home Secretary on 8th May, 1900. Dr. Turner said:—"The head wharfinger informs me that rats have not been numerous about the wharves. Notwithstanding this, about a fortnight ago he observed (what he had never observed before) four or five dead rats lying about the wharves every morning for three consecutive days; after this he found two rats daily, the last being found five days ago. The men at work informed him that they had observed rats, during the daytime, which appeared very dull and heavy and were very slow in their movements. He is quite positive that during the period no poison had been laid down at the wharves. The Inspector of Nuisances of the Rockhampton Municipal Council stated that during the past fortnight three or four dead rats had been observed every morning in a store opposite the wharves. He is positive that no poison had been laid down there."

The examination of four dead rats found in the store mentioned was not conclusive, the carcasses being too far advanced in putrefaction for reliable bacteriological examination.

The above facts, considered in conjunction with the entire absence of mediate contagion, induced Dr. Turner to believe that rats were the source of infection.

On the 20th April, 1900, a waiter, employed at the Criterion Hotel, Rockhampton, on getting up in the morning, felt a soreness in the left groin and noticed a lump there. He kept to his work until 27th April, though the swelling gradually got larger and was accompanied with pain in the groin. On 30th April he sought medical aid, when he was found to be suffering from a swelling and pain in the left groin and a temperature of 103-4 degrees Fahr. On 6th May, when he was examined by Dr. Turner, the mass formed by the left femoral glands was undergoing resolution, and signs of active inflammation had subsided. The swollen gland was punctured with a needle, and smear preparations from the fluid obtained showed on subsequent microscopical examination a few plague bacilli. Several smear preparations made by Dr. Voss a few days previously were also examined by Dr. Turner, and found to contain "very numerous plague bacilli." The above patient was in the habit of boarding all passenger steamers arriving at Rockhampton from Sydney, in his capacity of head waiter at the Criterion Hotel.

Up to 15th June, 1900, twenty-five cases had occurred at Rockhampton, and it had then been well established that an epizoötic existed among the rats of the city. Dr. Turner visited and reported upon the presumed place of origin of each of the cases. In no fewer than seventeen instances he observed conditions distinctly attractive to rats; the back yards, &c., being littered with kitchen scraps and waste food material. Five cases occurred at produce stores, one at a butcher's shop, and eight on premises where fowls and poultry were kept.

On the 27th April, 1900, a steward on board the ss. "Cintra" was reported by the Health Officer at Townsville to be suffering with symptoms of plague. The diagnosis was subsequently confirmed by Dr. Turner, smear preparations of the affected gland showing numerous plague bacilli. The patient had been taken ill on the 20th April, with intense headache, giddiness, vomiting, and epistaxis. It is a significant fact that the vessel left Sydney for Queensland ports on the exact day of the onset of his illness—viz., the 20th April. The case was isolated at the Quarantine Station, Magnetic Island, Townsville. Apparently his symptoms were not sufficiently alarming to attract the attention of the health authorities at Brisbane during the stay of the ss. "Cintra" at the latter port about the 22nd and 23rd April. The patient did not go ashore at Brisbane, and it was not till the 27th April that the first case among human beings was reported at Brisbane.

It was a singular coincidence that the first case of plague in man in Brisbane and the first case in Sydney should both have been vandrivers or carters, employed in removing goods from the wharves where dead and plague-infected rats were subsequently found. The first patient in Brisbane was reported on the 27th April, the diagnosis being confirmed by clinical and bacteriological evidence of such a convincing description as to place the diagnosis beyond any doubt. The patient was at once isolated, and all the necessary precautions taken to prevent the spread of the disease. How long an interval elapsed between the importation of the infection and the first case in man cannot be positively answered unless it be assumed that the plague-infected rat found on the 5th March, near the wharves, marked the first incidence of the disease in Brisbane. For about a fortnight previous to the above date rats had been collected by the officers of the local authorities and examined by the Government Bacteriologist, but with negative results. Though it is known that plague may assume an atypical clinical form at the outset of an epidemic, there was no reason to believe that any previous case had occurred in human beings in Brisbane, even of the so-called "pestis minor" type.

Of the first twenty cases at Brisbane no less than fourteen were connected with the shipping or the wharves, or lived in close proximity to the latter. Three of the cases were wharf labourers; three were carters engaged in removing goods from the wharves; two were storemen employed at the same city warehouses; one a grocer and produce dealer; two employed in city stores; one out of work; and two were stewards on vessels which had lately arrived at Brisbane from Sydney.

Inquiry of wharfingers and officials whose business took them to the wharves elicited the information that an unusual fatality among rats had taken place prior to the occurrence of the first case in man on 27th April. The disease continued to appear in isolated cases at Brisbane up to December, the last case being reported on 13th December, 1900. In all, fifty-six cases, with twenty-five deaths from plague, were recorded. From the wharves as a primary focus the infection extended along the main sewer lines to the city, with secondary indigenous foci on branches or reticulations connected with the main channel.

The above facts support the opinion that the disease was introduced by plague-infected rats from Sydney, and that the infection spread to the local rats, which in turn communicated it to man. Infectivity attached to locality, but in few instances to premises. Infection was not diffused by any of the usual modes of spread as in other infectious diseases—*e.g.*, influenza. The epidemic was not maintained by direct or mediate communication from the sick.

A glance at the spot map of the Brisbane area (Map A) will suffice to show that the infection was in some way or other connected with locality. Of the 300 cases which occurred in the Metropolitan Area during the eight epidemic years, 1900-1907 inclusive, no less than 210 were situated and successively occurred within a radius of one mile of the General Post Office situated in Queen street, the principal thoroughfare of the capital.

There was no evidence that human beings were communicating the disease one to another directly, nor by interchange of any goods, clothing, or other articles. It was seldom the disease attacked two persons in the same house, and the dwellings were separate and detached. The one circumstance common to them all was the actual discovery of plague-infected or dead rats either upon or in close proximity to premises in which some person fell sick with plague. The wandering proclivities of these particular animals eminently adapt them to act as carriers of infection, and by the exclusion of other means they must be regarded as the agency at work.

It would appear, therefore, that rats were the agents most directly involved both in the introduction and the dissemination of infection in these particular places. The area mentioned above includes the wharves and river frontages, which, early in 1900, were infested with rats, many of them found to be plague-infected. Assuming infection to have been imported from some plague-infected port oversea, or within the Commonwealth, and the wharves as the primary foci, extension to the areas indicated below could easily have taken place along the main sewer lines. There can be little doubt that the sewers (many were constructed in the early days of Brisbane and forgotten—the local authority having kept no plans or records of the same) constituted highways along which infected rats migrated. I may briefly advert here to a few incidental facts which bear upon the matter.

In a "Report on Plague in Brisbane, 1901-2," Dr. Halford (late Medical Officer to the Metropolitan Joint Board) states:—"There is an old sewer which was once an open one, built of brick many years ago, and more recently converted into a covered drain, which collects the drainage from a considerable area of Spring Hill and Wickham Terrace. There are many imperfections in this sewer, and a number of untrapped connections. It empties into the Brisbane River under Collin's Wharf at Petrie's Bight. Cases of plague have occurred along the course of this sewer, and, though their chronological order does not show it, there is a complete chain of cases commencing in the neighbourhood of the wharf and the embouchure of the sewer, and including those along its course to the district in which the drain commences. It is likely, however, that the communities of rats along the course of the sewer were infected from the wharf rats entering this highway, and, from the resulting local diffusion, cases among human beings occurred in the neighbourhood. . . . . On the Milton side of Brisbane, leading from the lower part of Petrie Terrace to the river near Boundary street, is a very old and defective sewer. Cases have occurred on the river road near where this sewer discharges, and there is a continuous chain of cases through Petrie Terrace to the furthermost part of Red Hill and parts of Paddington. . . . . In this way the extension along these defective sewers has accounted for the concentration of cases in Spring Hill and Petrie Terrace."

It will be shown later that the areas over which the epizoötic extended coincided with the areas over which the epidemic extended.

The following table shows the incidence of plague on man and on rats in the municipality for the years 1900-1907:—

Wards, Etc.	1900.		1901.		1902.		1903.		1904.		1905.		1906.		1907.		Totals.	
	Man.	Rat.	Man.	Rat.	Man.	Rat.	Man.	Rat.	Man.	Rat.	Man.	Rat.	Man.	Rat.	Man.	Rat.	Man.	Rat.
East Ward	11	19	4	31	15	30	5	26	5	46	1	30	3	19	9	8	53	209
West ,, ...	12	30	11	26	27	35	5	39	12	147	8	66	3	22	3	12	82	377
North ,, ...	13	22	7	14	9	14	3	3	1	12	5	1	1	3	0	1	39	70
Kangaroo Point Ward	2	1	0	1	0	0	0	1	0	0	3	2	0	0	0	0	5	5
Cintra Ward	1	0	2	0	4	0	0	0	0	0	0	0	0	0	1	0	8	0
Merthyr ,, ...	2	0	1	0	0	0	3	0	2	0	2	1	0	0	11	0	27	1
Valley ,, ...	5	11	2	16	6	5	2	10	0	18	1	14	0	3	11	0	20	77
Totals, City of Brisbane ..	46	83	27	88	61	84	18	79	20	223	20	114	7	47	35	21	234	739
South Brisbane	5	5	0	7	11	11	0	3	5	143	3	7	1	1	0	1	25	178
Other Local Authorities ...	5	2	9	6	10	11	3	2	5	14	5	8	3	0	1	0	41	43
Totals, Metropolitan Area	56	90	36	101	82	106	21	84	30	380	28	129	11	48	36	22	300	960

II.—THE EPIDEMIC.

General Review, 1900-1907.

1900.

FIRST EPIDEMIC.

The characteristic features of the epidemic were its duration and the number of places in the State affected. The first plague-infected rat was found at a Brisbane wharf on 5th March, 1900. The first human case of plague was reported on 15th April, 1900, at Rockhampton. The first case in man in Brisbane occurred on 27th April, followed by a rapid succession of cases comprising the epidemic.

The last case of plague in man was reported at Brisbane on the 13th December, 1900; the last infected rat at Brisbane on 24th October, 1900.

Of the outbreaks which occurred outside Brisbane (Metropolitan Area), the most serious was that of Rockhampton, where thirty-six cases with twenty-one deaths were reported.

The following statement shows the progress of the epidemic in Queensland during the year 1900:—

No.	Place.	Population.	Duration of Epidemic.	Cases.	Deaths.	Case Mortality.	Probable Source of Infection.
						Per cent.	
1	Brisbane (Met. Area)	125,500	27th April—13th December	56	25	44·6	Sydney
2	Ipswich	15,246	21st May	1	0	Nil.	Brisbane
3	Rockhampton ...	19,691	15th April—17th August ...	36	21	58·3	Sydney
4	Townsville	15,506	28th April—16th September	37	9	24·3	Sydney or Rockhampton
5	Charters Towers ...	20,976	30th September	1 (Pestis Minor)	0	Nil.	Townsville
6	Cairns	3,467	10th May—26th July ...	5	2	40	Sydney, Brisbane, or Townsville
			Totals	136	57	41·9	

Total recorded cases, 136. Deaths, 57. Case mortality, 41·9 per cent. Of the above cases, 9 were Chinese, 8 of whom died.

The corrected case mortality for whites was, therefore, 38·09 per cent.

The Type and Type-mortality.

Cases: Bubonic, 127; septicæmic, 5; pneumonic (secondary), 4.
Deaths: Bubonic, 50; septicæmic, 5; pneumonic (secondary), 2.

Sex Incidence: Males, 114; Females, 22. *Sex Mortality:* Males, 47; Females, 10.

The case and death incidence of 97 cases where age was noted was:—

Age Group.	-5.	5-9.	10-14.	15-19.	20-24.	25-29.	30-34.	35-39.	40-44.	45-49.	50-54.	55-60.	Over 60.
Cases ...	4	10	8	4	19	15	7	8	6	7	4	2	3
Deaths...	3	5	4	1	8	5	1	3	3	3	3	1	2

BRISBANE (METROPOLITAN AREA).

The following statement gives the figures for the epidemic and the epizoötic month by month:—

January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
						MAN.						
0	0	0	3	11	8	15	4	5	4	4	2	56
						RATS.						
0	0	5	3	0	15	21	24	15	7	0	0	90

Number of rats examined	735.
,, ,, infected	90.
Percentage of rats infected ...	12·2 per cent.

1901.

SECOND EPIDEMIC.

After an interval of seventy-five days, plague again made its appearance in Brisbane; the first case, that of a young man employed at the Meatworks, Eagle Farm, was reported on the last day of February, 1901.

During the month of March, seven additional cases were notified in Brisbane. Scattered cases in the city and suburbs continued to occur till the end of July, when the sequence of cases terminated. A sporadic case was reported in August, another in October, and one case in December.

In all, 36 cases with 12 deaths were recorded, a case mortality of 33·3 per cent. Of these, 27 were males with 11 deaths, and 9 females with 1 death. One male Chinese recovered.

It is an interesting fact that no case of plague was officially reported outside of the Brisbane Area for this year. The sporadic character of the cases was evident from the small proportion of the population affected and the time periods of the attacks. In five cases abdominal, and in nineteen cases cerebral, symptoms were pronounced. In one fatal septicæmic case secondary pneumonic symptoms were present. There was no spread from case to case.

The highest number of cases reported in any one week was four, for the week ending 14th May, 1901.

A larger proportion of rats was examined than in the previous year. Of 1,851 rats examined at the Bacteriological Institute, 101, or 5·45 per cent., were found to be plague-infected. The curve of rat-plague for 1901 reached its summit in June; that of human plague in May. No infected rats were found during the months of January, February, and March; the heaviest incidence falling on the months of April, May, June, and July. From July to the end of the year, the percentage of plague-infected rats remained low, but it is significant that a considerable rise in mortality and infection was noted during the earlier months of the following year.

The figures for the months during which the epidemic and epizoötic lasted are as follows:—

January.	February.	March.	April.	May.	June	July.	August.	September.	October.	November.	December.	Total.
						MAN.						
0	1	7	7	12	3	3	1	0	1	0	1	36
						RATS.						
0	0	0	16	23	32	20	8	1	0	1	0	101

Date of first human case, 28th February, 1901. Date of last human case, 9th December, 1901.
First infected rat found, 4th April, 1901. Last infected rat found, 22nd November, 1901.

SITUATION OF BUBOES.

——	Right Femoral.	Left Femoral.	Right Inguinal.	Left Inguinal.
Male	3	14	3	1
Female	4	3	3	4
Total	7	17	6	5

1902.

THIRD EPIDEMIC.

During the year now under review, the epidemic in Brisbane ran a rapid and somewhat severe course, reaching its maximum in the month of April. By the end of May, 81 cases had been recorded, with 26 deaths, or a mortality of 32·09 per cent. for the Metropolitan Area. One sporadic case occurred in August after a quiescent period of over two months.

About 10 per cent. of all rats captured or found dead in the Metropolitan Area was bacteriologically examined. Of 3,851 rats examined, 106, or 2·75 per cent., were found plague-infected. The epizoötic also reached its maximum in April; and it will be seen from the following comparative statement that the monthly incidence on rats bore a remarkable coincident relationship to the cases in man. During the second half of the year there was a striking absence of cases both in man and rats.

METROPOLITAN AREA.

Table showing monthly incidence on man and rats:—

January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
						MAN.						
1	13	21	28	18	0	0	1	0	0	0	0	82
						RATS.						
3	6	7	53	16	12	8	1	0	0	0	0	106

COURSE OF OUTBREAKS IN TIME PERIODS, 1901 AND 1902.

—	1901.		1902.	
	Rats.	Man.	Rats.	Man.
January	0	0	3	1
February—March	0	8	13	34
April—May	39	19	69	46
June—July	52	6	20	0
August—December	10	3	1	1
Total	101	36	106	82

Seven cases were reported from Townsville, 1 from Bundaberg, and 1 from Gladstone.

The subjoined tabular statement shows the incidence of plague in the State of Queensland for the year 1902:—

Place.	Date.	Cases.	Deaths.	Case Mortality.
				Per cent.
Brisbane	27 January to 4 August	82	26	31·7
Townsville	20 August to 26 November	7	5	71·4
Bundaberg	13 October	1	1	100
Gladstone	25 August	1	1	100
		91	33	36·2

Three cases were Chinese ; all fatal. Corrected death rate for whites, 34.09 per cent.

Sex Incidence: Males, 63; females, 28—total, 91. *Sex mortality:* Males, 19; females, 14—total, 33.

The following statement gives the case and death incidence for 90 cases where age noted:—

Age Group.	-5.	-10.	-15.	-20.	-25.	-30.	-35.	-40.	-45.	-50.	-55.	-60.	Over 60.
Cases ...	1	3	13	14	12	10	9	5	9	7	2	2	3
Deaths...	1	0	2	4	3	4	3	3	3	5	1	2	2

With the exception of five, all the cases were primarily bubonic in type. In 32 cases, cerebral; and in 4 cases, abdominal symptoms were marked. Two cases had primary pulmonary symptoms, a cover-glass preparation from the lungs in one case after death showing numerous *B. pestis*.

SITUATION OF BUBOES.

(84 cases noted in whole State.)

—	Right Femoral.	Left Femoral.	Right Inguinal.	Left Inguinal.
Males	19	15	14	19
Females	5	6	4	2
Total	24	21	18	21

3 cases had femoral buboes (side unstated).
2 cases had inguinal buboes (side unstated).
In 2 *post-mortems* the mesenteric glands were enlarged and hæmorrhagic.

1903.

FOURTH EPIDEMIC.

The outbreak, the fourth in Queensland, consisted of 29 cases with 17 deaths, distributed as follows:—

Place.	Date.	Cases.	Deaths.	Case Mortality.	Source of Infection.
				Per cent.	
Brisbane ...	8 February—11 September	21	11	52·3	Plague-infected rats.
Bundaberg ...	...	2	1	50·0	ditto.
Rockhampton	11 February—21 February ...	2	2	100·0	ditto.
Townsville ...	...	3	2	66·6	ditto.
Cairns ...	...	1	1	100·0	ditto.
Total ...	...	29	17	58·6 per cent.	

The gross mortality was thus 58·6 per cent. Four of the cases were Chinese, all fatal, and if these be deducted, the fatality amongst whites was 52 per cent. Several cases were only discovered by *post-mortem* examination.

Sex Incidence: Males—Cases, 22; deaths, 15. Females—Cases, 7; deaths, 2.

Age Incidence: The observed incidence was on ages from 5 years to 55, 18 cases occurring at ages between 15 to 35:—

Age Groups.	-5.	-10.	-15.	-20.	-25.	-30.	-35.	-40.	-45.	-50.	-55.	-60.	Total.
Attacks	1	2	4	4	3	3	4	3	2	2	1	0	29
Deaths...	1	1	2	3	2	2	2	2	1	0	1	0	17

All cases were bubonic in type, variation in the type of the disease being more carefully watched for than variation in severity of attack.

Situation of Buboes.—Right femoral, 3; left femoral, 5; right inguinal, 5; left inguinal, 10; right axillary, 2; left axillary, 1; right cervical, 1. Two cases had multiple buboes.

Every case was definitely proved by clinical and bacteriological evidence of a convincing character. A large number of suspected cases were negatived after careful consideration and investigation. Segregation of "contacts" in an isolation building was discontinued, except in cases where pneumonic manifestations were present. The usual precautions as to isolation of patients and disinfection of infected premises were observed. A general cleansing of the city and rat destruction on a large scale were carried out, the permanent departmental gang being increased by the addition of 100 men for several weeks. Rat destruction was continued throughout the year, the Government maintaining rat-catchers in every important centre throughout the State. As in former years, the epizoötic preceded and accompanied the epidemic. No unusual mortality from plague was observed in the rats. With the exception of rats and mice, the influence of animals in the spread of the disease was inappreciable. The importation of ship-borne rats and inter-communication between shore and ship rats was prevented, as far as it was possible to do so, by the stringent enforcement of the plague regulations relating to the mooring of vessels while in port.

Infected rats were found at Cairns, Townsville, Rockhampton, Bundaberg, and Brisbane. The outbreak in Brisbane began on 18th February, 1903, and ended on 23rd May; two sporadic cases appearing in September. In all, 21 cases were reported from the Metropolitan Area; the epidemic furnishing a smaller number of cases than in any previous year.

Both infected premises and infected rats were confined to a much smaller area—practically the city proper—than in the previous outbreaks. The presence of infected rats on premises where indigenous cases had occurred was demonstrated often enough to support the now generally accepted position, "that the danger of contracting plague stands in relation to the presence of rats in dwellings and enclosed premises." Plague-infected rats, however, were found on many premises where no case of human plague occurred, but in one of the earlier cases some 200 or more dead carcasses of rats (bodies too decomposed for examination) were removed from beneath the floors of a granary in close proximity to a house from which a plague patient had been removed a short while before.

Of 134 mice collected and examined, 4 were found to be plague-infected.

Of 16,409 rats collected in the Metropolitan Area, 14,227 were examined bacteriologically, and 84 were found to be infected—a percentage of 0·5.

The subjoined table shows the monthly incidence of plague on man and rats in the Metropolitan Area, 1903:—

January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
						MAN.						
0	5	4	6	4	0	0	0	2	0	0	0	21
						RATS.						
1	*8	*30	20	15	6	0	0	4	0	0	0	84

* One infected mouse each month included.

A large proportion of the infected rats caught in the latter part of the year came from the city wharves.

1904.

FIFTH EPIDEMIC.

During the month of February, plague again appeared at Brisbane, and continued to manifest itself up to August, when it once more for the time subsided.

The last case of human plague in Brisbane in 1903 was on 11th September. The last infected rat in 1903 was found on 28th September. The first infected rat of 1904 was found on 8th January. The first case of human plague in 1904 was reported on 9th February. The fifth outbreak in Queensland was marked by 35 cases with 12 deaths, distributed as under:—

Place.	Date.	Cases.	Deaths.	Case Mortality.
				Per cent.
Brisbane	9th February—14th September ...	30	8	26·6
Ipswich	10th July	1	1	100
Maryborough	3rd June—13th	2	1	50
Cairns	16th February—22nd March ...	2	2	100
	Total	35	12	34·2

Gross case mortality, 34·2 per cent. Of the 12 fatal cases, 2 were Chinese, 1 a kanaka, and 1 an aboriginal. Corrected case mortality for white races, 25·8 per cent.

Sex Incidence and Fatality.—The subjoined table shows that but one death (an aboriginal) occurred among females within all the infected areas:—

—	Brisbane.		Ipswich.		Cairns.		Maryborough.		Total.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Cases	25	5	1	0	1	1	2	0	29	6
Deaths	8	0	1	0	1	1	1	0	11	1

Endemic maintenance and dissemination of the disease by rats were the main causes of the outbreak for the year under discussion.

During the period, January to December, 46,056 rats and 27,948 mice were destroyed; of these, 25,653 rats and mice were examined at the Bacteriological Institute, with the following results:—

Rats examined	20,731
Rats infected	401
Percentage of rats infected	1·4
Mice examined	4,963
Mice infected	3
Percentage of mice infected	0·06

The above figures include infected rats found at Ipswich and Maryborough.

Of the 401 infected rats, 78 were obtained from the "infected area" in the City of Brisbane, and 88 were found during the cleansing operations in one produce store in the South Brisbane Area.

Species of Infected Rats.

Mus decumanus	309
Mus Alexandrinus	32
Mus rattus	31
Undetermined	8
Total	401

Wherever infected rats were found, the particular and all neighbouring premises were thoroughly searched for rats, and all sanitary defects brought under the notice of the local authority concerned.

The incidence of plague both in man and rats was heaviest on particular localities where produce and grain were stored.

Most of the patients were men in adult life and associated directly or indirectly with the produce industry or other food trades.

Of the 30 persons who suffered from plague within the Metropolitan Area in 1904, 9 were produce-store employees, 3 were engaged in stable duties, 2 were grocers, and 1 a cook in an oyster saloon.

Of the 401 infected rats found in business premises in the Metropolitan Area, no less than 130 were obtained from produce stores, 25 from grocers' stores, 19 from hotels, and 10 from butchers' shops.

The continued incidence of plague cases and plague-infected rats on produce stores led to the issuing of instructions by the Commissioner early in the year for a systematic inspection and cleansing of all produce and grain stores within the Metropolitan Area.

In 30 stores so cleansed and disinfected, 541 dead rats and 957 live rats, 305 dead mice and 607 live mice, were discovered.

Of the 1,498 rats, 109 (7·3 per cent.) were plague-infected; and of the 912 mice, 2 (·22 per cent.) were found to be plague-infected.

On 22nd August the Commissioner declared an "Emergency" in the South Brisbane Area, under section 20 of the Health Act, and a thorough cleansing of this area was carried out, the work occupying one month.

All the 35 cases were primarily bubonic, 4 becoming septicæmic.

The subjoined table shows the situation of buboes in the 35 cases:—

—	Left Femoral.	Right Femoral.	Right Inguinal.	Left Sternomas.	Right Axillary.	Left Epitroch.	Left Inguinal and Right Femoral.	Right Inguinal and Left Femoral.	Right Axillary and Left Femoral.
Males ...	18	4	3	1	0	1	1	1	1
Females ...	2	1	1	0	1	0	0	0	0
Total ...	20	5	4	1	1	1	1	1	1

Four patients succumbed to virulent general hæmal infection. There was no case of primary plague-pneumonia. Forty-three cases reported as suspicious were negatived on clinical and bacterioscopical examination.

The following figures show the age incidence for 35 cases:—

Age Group.	-5.	-10.	-15.	-20.	-25.	-30.	-35.	-40.	-45.	-50.	-55.	-60.	Total.
Cases ...	0	2	3	7	4	4	5	5	2	1	0	2	35
Deaths...	0	0	2	2	1	1	2	1	1	1	0	1	12

METROPOLITAN AREA, 1904.

The progress of the disease and the incidence in the several months on man and rodents may be followed in the subjoined tabular statement:—

January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
						MAN.						
0	5	0	7	11	1	0	5	1	0	0	0	30
						RATS.						
21	3	25	25	*89	150	38	17	4	0	6	2	380

* Three infected mice are included in May.

1905.

SIXTH EPIDEMIC.

Nearly four months elapsed between the last human case of 1904 and the first of 1905, which latter was reported in Brisbane on New Year's Day, 1905.

The last case of plague in human beings in Brisbane in 1904 was reported on 14th September. The last infected rat of 1904 was found on 15th December.

The first case in man in 1905 was discovered on 1st day of January; the first infected rat of 1905 was found on 4th January.

Twenty-eight cases occurred in Brisbane; 1 at Bundaberg; 8 at Ipswich; 1 at Childers; 2 at Cairns; 10 at Maryborough; and 6 cases, all fatal, at Townsville; a total of 56 cases with 33 deaths. The gross death rate was, therefore, 58·9 per cent. Deducting two fatal cases among Chinese and one, an aboriginal, the figures give a corrected death rate for whites of 56·6 per cent.

Several fatal cases were not treated for plague—*i.e.*, cases diagnosed *post mortem*, or dying before treatment was possible. The death rate for treated cases was 42·1 per cent.

As in former years, the epidemic in Brisbane was markedly periodic. The procedure adopted with regard to the examination of persons reported by medical practitioners to be suffering from plague did not differ from that of former years. All cases accepted as "Positive" satisfied both clinical and bacteriological investigation as well as inoculation experiments in the laboratory. Consequent on the results obtained by Dr. Hunter, of Hong Kong, the blood of a large number of patients was examined in the early stages of the disease for the B. Pestis. Of 8 cases in which plague bacilli were found in the blood, 4 terminated fatally. Cultural, feeding, and other experiments conducted with such suspected material as grain, fodder, produce, and the like gave negative results.

The total number of cases in the State for the year 1905 was distributed as follows:—

Place.	Dates.	Cases.	Deaths.	Case Mortality.	Source of Infection.
				Per cent.	
Brisbane	January to June	28	15	53·5	Plague-infected rats
Bundaberg	3rd February	1	1	100	Ditto.
Ipswich	5th May to 7th May ...	7	1	14·3	Ditto.
Ditto.	4th July	1	1	100	Ditto.
Childers	16th May	1	1	100	Infected fodder from Bundaberg
Maryborough	19th May to 10th June ...	10	8	80	Plague-infected rats
Cairns	24th June	2	0	0	Ditto.
Townsville	August to October ...	6	6	100	Ditto.
Totals	...	56	33	58·9	

On the 3rd February, a man, aged twenty-six years, died at Woongarra, Bundaberg. *Post-mortem* examination and bacteriological investigation of specimens proved his case to have been one of bubonic plague.

On the 4th May, a case of plague was discovered at Ipswich. Within a week six other cases occurred. Six of the seven patients were removed to the "Colmslie" (Brisbane) Plague Hospital, and all of them recovered. The remaining case, a boy, aged eight years, being too ill for removal at the time, was attended at his own home at Ipswich, and died there on the 9th May.

All the Ipswich cases were bubonic in type, and were traced directly to a produce store, a dilapidated shed in which rats had been reported to be dying in large numbers a week before the outbreak. The store was demolished, much of the contents destroyed by fire, and the rest disinfected. A large number of rats were destroyed in the Ipswich Area, and every precaution was promptly and energetically carried out. As a result, the outbreak was limited to these seven cases, and no further development took place. (*See* "Sub-Epidemics.")

On the 16th May, a man died of septicæmic plague at Childers, a township with a population of about 2,000, and situated 39 miles from Bundaberg, and 41 miles from Maryborough. Large numbers of rats were found dead in the town, the epizoötic being severe. Circumstances seemed to indicate that infection was conveyed by fodder from Bundaberg. As a precautionary measure, the whole town was inspected, produce stores, &c., put in order, insanitary conditions remedied, and a special staff of men employed in rat-destruction. Similar precautions were carried out at Bundaberg. No further cases occurred at Childers.

A series of cases, with pneumonic manifestations, occurred towards the end of May and the beginning of June at Maryborough, a city of some 12,000 inhabitants, situated on the Mary River, about 20 miles from its entrance, and distant 167 miles by rail from Brisbane.

On the 4th June, an official inquiry was held by the Commissioner of Public Health, at the Municipal Chambers, Maryborough, into the circumstances of the outbreak of plague in that city. The outbreak was characterised by pneumonic manifestations, without distinct enlargement of the lymphatic glands, a type of plague which, fortunately, has been rare in Queensland. The members of one family (O'C.), consisting of seven children, residing with their father at Newtown, Maryborough, in a state of great poverty and neglect, were seized within a few days of each other with acute gastro-intestinal symptoms, which terminated in five of the cases with symptoms of pneumonia. The first case, a boy of seventeen years, died on 25th May, and prior to his death he was attended at his own home by a local medical practitioner, who diagnosed the case as one of "pneumonia." On the 28th May, a neighbour of the above family (O'C.), a Mrs. E., who had visited and assisted in nursing the above case, sickened with headache, vomiting, &c., and died at her home on the 31st May. In this instance a certificate of death was refused by the local medical practitioner, and a *post-mortem* examination was ordered by the Commissioner. The body, however, was buried without a death or other certificate by the Maryborough cemetery authorities. Four other children of the O'C. family, who had sickened with symptoms similar to the first case, were sent into the General Hospital by a medical practitioner on the 28th May.

On the 31st May, a girl (E. O'C.), aged seven years, and a boy (Jas. O'C.), fifteen years, died at the General Hospital with symptoms of "broncho-pneumonia," and the medical superintendent signed the death certificate to that effect. On the 2nd June another member of the O'C. family, a boy (R. O'C.), aged ten years, and on the 3rd June, a girl (M. O'C.), aged three and a-half years, died at the hospital, both with symptoms of plague-pneumonia.

Two staff nurses of the Maryborough General Hospital, who had attended the above patients from the time of their admission to the wards, were taken ill with symptoms of pneumonia, and died on 6th and 12th June respectively. A third nurse who had sickened recovered.

The above is a short history of the association and distribution of the Maryborough cases. Apparently suspicion was not aroused until the coincident deaths of Mrs. E., at her residence, and the two children in the General Hospital on the one day—viz., 31st May. The nurses and inmates of the General Hospital were thus exposed to infection before the real nature of the disease had been discovered and the Department communicated with. A rapid development took place, five deaths occurring at the hospital within six days. In all, ten cases occurred, of which eight terminated fatally.

It is to be regretted that the precautionary measures taken by the Department of Public Health, and which were carried out with such signal success at a later date, could not have been enforced earlier. The Department was not made aware of the occurrence of several sudden deaths from some suspicious but undetermined cause till after the events had happened.

The measures taken by the Department for the suppression of the outbreak were preventive inoculation of doctors and nurses in attendance, together with the wearing of specially designed costumes and respirators; isolation of the sick, segregation of those exposed to infection, destruction by burning of the infected house with its contents, quarantine of the hospital premises, disinfection of clothes, disinfection and evacuation of premises, including the General Hospital, extensive cleansing operations, and the destruction and examination of rats caught in the town. His Worship the Mayor and the Council of the city of Maryborough afforded active and willing co-operation. No further development occurred. A special report on the outbreak was prepared and presented to the Honourable the Home Secretary, who authorised its publication. (*See* Appendix E, Part II., also "Localised Epidemics.")

On the 4th July, a fatal case of bubonic plague was reported from Ipswich. Forty-seven rats and three mice obtained from Ipswich were examined at the Bacteriolcgical Institute, and found free from infection.

A mild case, a man employed as a rat-catcher, was reported from Cairns during the month of June. Early in this month two dead rats had been found at Cairns, with naked-eye evidence of plague.

A fatal case of bubonic plague occurred at Townsville on the 4th August, 1905. The patient, a boy, lived in a coal hulk moored at the jetty wharf. Sixty-three plague-infected rats were discovered at this wharf during the previous month.

During the month of September, three further cases were reported, and on the 15th October an aboriginal admitted to hospital died of plague on 21st idem. A further fatal case was reported on the 19th October. In all, there were six cases and six deaths—a gross mortality of 100 per cent.

Of 489 rats examined at Townsville during October, five were found plague-infected. The last infected rat was found there on the 6th October.

The following table gives the type of the disease for the year 1905:—

Place.	Cases.	Bubonic.	Septicæmic.	Pneumonic.	Deaths.
Brisbane (Metropolitan Area) ...	28	24	4	...	15
Bundaberg	1	1	...	...	1
Ipswich	8	8	...	...	2
Childers	1	...	1	...	1
Maryborough	10	1	6	3	8
Cairns	2	2	...	...	...
Townsville	6	5	1	...	6
Total	56	41	12	3	33

The following figures show the sex incidence and mortality:—

Cases: Males, 38; females, 18—total, 56. Deaths: Males, 21; females, 12—total, 33.

The subjoined table gives the number of attacks and deaths under age groups of fifty-six cases:—

Age Group.	-5.	-10.	-15.	-20.	-25.	-30.	-35.	-40.	-45.	-50.	-55.	-60.	Over 60.	Total.
Cases ...	1	3	8	11	7	10	5	6	2	2	0	1	0	56
Deaths	1	3	5	7	4	6	2	4	0	1	0	0	0	33

The monthly incidence of plague in the Metropolitan Area for the year 1905 was as follows:—

January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
						MAN.						
4	16	2	1	4	1	0	0	0	0	0	0	28
						RATS.						
5	*59	48	3	7	5	1	0	0	0	1	0	129

* One infected mouse is included in February.

The last case of plague in human beings was reported on the 14th June. The last infected rat was found on 29th November, 1905.

With the exception of one infected rat on the 4th July, and one on the 29th of November, there was no plague in man or rats from July to December.

The total number of rats and mice destroyed, examined, and infected within the Metropolitan Area for the year 1905 was as follows:—

1905.	No. of Men Employed on Gang.	No. of Rodents Destroyed	No. of Rodents Examined.	No. of Rodents Infected.	Percentage of Infected Rodents.
January to June	27	19,382	10,101	127	0·77
July to December	20	11,993	6,679	2	
Totals	...	31,375	16,780	129	...

The monthly returns were:—

1905.	Rats Examined.	Mice Examined.	Found Plague Infected.
January	1,242	330	5
February	1,669	518	59
March	1,703	514	48
April	1,218	313	3
May	1,424	342	7
June	549	249	5
July	697	195	1
August	753	182	0
September	1,102	280	0
October	1,035	235	0
November	845	174	1
December	1,016	165	0
Total	13,283	3,497	129
	16,780		

Species of Infected Rats.

Mus decumanus	87
Mus rattus	40
Mus Alexandrinus	1
Mus musculus	1
	129

Situation of Buboes.—Right femoral, 12; left femoral, 10; right inguinal, 3; left inguinal, 3; right axillary, 1; left axillary, 3; right cervical, 1; left cervical, 1. Double buboes—right femoral and right inguinal, 3; left femoral and left inguinal, 2; multiple buboes, 4; mesenteric glands, 1. No buboes, 12.

1906.

SEVENTH EPIDEMIC.

As in previous years, plague again made its appearance in Brisbane during the first half of the year, but its manifestation in the Metropolitan Area was exceedingly limited, the number of cases in man and rodents being the lowest on record since the introduction of the disease into Queensland in 1900.

The first case in man in 1906 was reported on the 6th March; the first plague-infected rat discovered on the 12th January.

The total number of cases for the Metropolitan Area was 11, 8 of which occurred in the first half of the year—March to June—4 cases being reported in the month of April.

After an interval of 151 clear days, two sporadic cases were reported late in November, and one case—reported after death—in the last week of December.

In four of the above cases the nature of the disease was revealed only after *post-mortem* examination, and before treatment could be made available, while a fifth fatal case was that of a Chinaman. The corrected death rate for whites was 55·5 per cent.

That the immunity from plague in the Metropolitan Area for the greater part of the year was the outcome of the constant and systematic efforts of the departmental rat-destruction gang there can be little doubt. The number of rats destroyed could easily have been doubled had the Department been given the assistance of the local governing bodies, individual householders, and occupiers of premises. An amendment of the Health Act in the direction of the making of occupiers responsible for keeping their properties free from infestation with rats was urgently recommended by the Commissioner, but no action was taken in this direction during the session of Parliament.

Though no cases of plague occurred within the Metropolitan Area until the 5th March, many "suspect" cases reported were negatived after due investigation, clinically and bacteriologically. One of these is worthy of remark. After an attack of influenza, the patient developed a large tense right femoral bubo, with high fever and delirium. "Filariasis" was diagnosed, and a dead worm was found in the blood.

Departmental procedure did not differ from that of former years except at two points—viz., a more systematic examination of rats caught at the ports other than that of Brisbane; and, secondly, in the destruction of rats by the employment of the rat virus of Danysz. The virulence of the Danysz bacillus was exalted and constantly tested, a litre of virulent broth being prepared at the Bacteriological Institute twice a week.

The monthly incidence of plague in the Metropolitan Area on man and rats was:—

January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
						MAN.						
0	0	2	2	2	2	0	0	0	0	2	1	11
						RATS.						
1	6	6	3	5	6	2	8	0	1	0	10	48

The total number of rats destroyed, examined, and found infected was:—

Year.	Number Killed.	Number Examined.	Number Infected.	Percentage of Infected Rodents.
1906	19,643	12,195	*48	0·39

* On the 9th and 10th February, 1906, five plague-infected rats were found on board the coastal steamer "Aramac," but these are not included in the Brisbane list of infected rats, owing to the fact that they were found on the steamer on her arrival direct from Sydney and before berthing at the Brisbane wharf.

The monthly rat returns were:—

1906.	January.	February.	March	April.	May.	June.	July	August.	September.	October.	November.	December.	Total.
Killed	1,850	1,982	2,640	2,250	2,594	2,340	708	1,016	580	806	1,039	1,842	19,647
Examined ...	1,031	1,203	1,488	1,312	1,448	1,461	567	718	364	522	795	1,286	12,195
Infected ...	1	6	6	3	5	6	2	8	0	1	0	10	48

Species of Infected Rats.

Mus rattus	31
Mus decumanus	16
Mus Alexandrinus	1
	48

After an absence of six years, plague in epidemic form reappeared at Rockhampton on 3rd April, 1907.

There is good reason to believe that the infection was imported by rats from vessels arriving at the town wharves.

In all, there were 11 cases and 4 deaths. Eight of the cases were directly connected with the one warehouse situated in East street, Rockhampton, and all 8 cases were reported within a period of 8 days.

On the 23rd April, the Commissioner of Public Health held an inquiry at Rockhampton into the circumstances of the outbreak.

It was ascertained that dead and diseased rats had been found in the warehouse premises just prior to the outbreak. Dead rats had also been found in a store adjacent to the warehouse. There was evidence to show that the first two cases, both of which were fatal, handled dead and sick rats prior to their attacks.

Three sporadic cases occurred at Rockhampton early in May, one patient being a rat-catcher employed by the Health Department. He was bitten on the hand by a rat on the 9th May, and was reported as a case of plague on the 13th idem.

Cleansing operations in the town were vigorously pursued, and a gang of seven men employed in rat-destruction. Of 195 rats examined at Rockhampton none were found plague-infected.

A case of bubonic plague was reported by the Health Officer at Cairns in the month of July, and six further cases were reported during September. Three cases also occurred in the month of October. In all, there were ten cases with one death.

The town of Cairns was thoroughly cleansed under the superintendence of the Chief Inspector of the Health Department, and a vigorous crusade carried on against rats. Although active search for rats was continued during and after the outbreak, comparatively few rats were found in the town. One rat, dead of plague, was found in the premises of case 2, ten days previous to the onset of illness.

The following statement shows the incidence of plague in the State of Queensland for the year 1906:—

Place.	Dates.	Cases.	Deaths.	Case Mortality.
				Per cent.
Brisbane	March—June	8 } 11	4 } 7	50·0 } 63·6
	November—December	3	3	100·0
Rockhampton	April—May	11	4	36·3
Cairns	July, September, October ...	10	1	10·0
	Total	32	12	37·5

Total cases include 1 Japanese and 1 Chinese.
Corrected death rate for Whites, 33·3.

Sex Incidence: Males, 25; females, 7—total, 32. *Sex Mortality:* Males, 12; females, 0—total, 12.

Age Incidence:—

Age Group.	-5.	-10.	-15.	-20.	-25.	-30.	-40.	-45.	-50.	-55.	-60.	Over 60.	Total.
Cases ...	0	2	7	2	3	4	7	5	1	0	0	1	32
Deaths...	0	0	4	0	3	3	0	0	1	0	0	1	12

Type of Disease: Bubonic, 27; septicæmic, 5; pneumonic, 0.

Situation of Buboes.—Right femoral, 9; left femoral, 5; right inguinal, 5; left inguinal, 1; right axillary, 4; left axillary, 3. No buboes. Septicæmic cases, 3.

1907.

EIGHTH EPIDEMIC.

For the eighth year in succession Brisbane was visited with an epidemic of plague. The last case in man in 1906 was reported on the 1[illegible]th December, the previous case on the 23rd November.

The last infected rat of 1906 was found in Brisbane on 28th December.

The first case in human beings in 1907 occurred at Paddington, a suburb of Brisbane, on 3rd January. The first plague-infected rat was found in a newspaper office in the city the day before—viz., the 2nd January, 1907.

The experience gained in former years was of service in the methods adopted for dealing with the actual epidemic, but it was felt that more efficient methods of destroying rats, and, if possible, preventing the recurrence of the disease year after year was the real aim and end to be kept in view.

Cases both in man and rats began early in the year. Some 21 cases in human beings were reported for the month of January, 1907, in the Metropolitan Area. Of the above, no less than 16 cases occurred in one small localised suburban area heretofore free of infection. These cases were interesting from the fact that all were attacked at about the same time, and, apparently, had one source of infection common to all. The first 6 cases were those of schoolboys at home for the holidays, and as playmates were in constant association with each other up to the time of their attacks. A brother of the first case, a boy of fifteen years, died in the Hospital for Sick Children, on the 13th January, with symptoms of cerebro-spinal meningitis. This death was rapidly followed by similar initial cerebral symptoms in five of his playmates, including his brother (case 1). It was not until after admission to the Children's Hospital, on 16th January, of these latter cases, and the subsequent development of glandular swellings, that plague was suspected as the real cause of the illnesses.

The father of the boy who died in the Children's Hospital with symptoms of cerebro-spinal meningitis, and of the first case, kept a produce, fruit, and general store in James street, Kingsholme, New Farm. He obtained the bulk produce from a produce store in the city where infected rats had been found on 23rd December, 1906. The abovementioned boys and their playmates were in the habit of frequenting the produce shed and assisting in the handling of the produce. There was evidence of recent rat infestation in the shed, a history of dead rats having been seen previous to the outbreak, while twelve skeletons of rats were discovered among the bags of produce and beneath the floor of the shed at the time of the cleansing operations. A great accumulation of filth and rubbish removed by the cleansing gang from beneath the general and fruit stores is mentioned below in connection with later cases.

During the stay of the earlier cases in hospital another schoolboy living in a house abutting on to the store premises was taken ill on 18th January. This boy had a cervical gland and a lesion on the lower lip. This patient had noticed a cat bringing dead rats from the incriminated store into the yard of his residence. He stated that the rats were very offensive, and that the cat which he handled had numerous fleas on its body. This boy died in the Plague Hospital on the 25th January. The grandmother of this boy lived with him and nursed him at the onset of his illness. She had been in the habit of visiting the general store for groceries. She died at her home on the 21st January. In the meantime, two other children living on the infected store premises had sickened, and were removed to the Plague Hospital.

A schoolboy, aged eight years, living with his parents in the immediate rear of the store, was also attacked about this time, and died at his own home, the case being reported after death. The *post-mortem* examination revealed the presence of *Bac. pestis.* Dead rats were also found on the premises of the latter case.

A man, aged forty years, residing immediately opposite the store premises, and who had been temporarily employed at the store, was also attacked. He had also been engaged in removing rubbish from beneath the floors of the latter prior to the outbreak among the first six schoolboys, two of his sons being among those attacked. A sister, aged fourteen years, of the latter was also attacked about the same time (22nd January) as her father, and both were removed to the Plague Hospital. The man died on the 23rd January; just before death he spat up some blood-stained sputum, which contained *B. pestis.* He and his three children lived in a dilapidated house standing on piles several feet below the level of the street. The space beneath the house was used as a living and sleeping room, and contained two beds, the mattresses of which were infested with fleas and bugs. The latter were burned and the house condemned as unfit for habitation.

Attention was now directed to three men employed by the Health Department in cleansing and disinfecting the "infected area." They fell sick about the 23rd January, and were reported on the 26th. One of the patients died in hospital on 31st January; the other two progressed slowly but finally recovered. These men had been employed in removing a large accumulation of filth, consisting of animal and vegetable matter, dust, and *débris* from beneath the incriminated general and fruit stores. These men, therefore, took ill before the cleansing operations had had time to become effective. The three men, with other members of the gang, had also worked at cleaning out the produce shed where it was noticed that fleas were particularly numerous, several of the men who remained well in health being severely bitten.

Compulsory and voluntary evacuation of the entire area, with disinfection and cleansing of every house in the locality, were measures adopted; the inhabitants segregated and not allowed to return until the cleansing operations were completed. The whole of the soil, sewage sodden and polluted with filth, in the vicinity of the store and other premises was thoroughly saturated with sulphuric and carbolic acids, the ground being dug up for a foot or so. The spaces—dark, dirty, and damp—beneath the floors, especially those of the general and fruit stores, were also cleansed and ventilated.

A further account of the New Farm cases will be found under the heading "Localised or Sub-Epidemics."

Many sanitary defects were remedied, and certain structural improvements ordered to be carried out. Several houses in the area were condemned as unfit for human habitation. The floor in the produce shed was ordered to be concreted, and a retaining-wall in front of the infective premises built and made rat-proof.

No further cases occurred in this area.

Sporadic cases continued to be reported from other parts of the Metropolitan District during the months of February, March, April, and May, *and in every instance the source of infection was traceable to rats.* In the majority of cases, plague-infected rats were not caught alive on the premises at the time of the cleansing operations, but dead rats, often too decomposed for examination, were invariably found on the premises, or, there was a distinct history of rats having died on or near the infected houses previous to the onset of the cases reported.

On the 5th April, a woman, aged twenty-three, was delivered of a full-term living child at the Lady Bowen Hospital, Brisbane. She had been admitted with a high temperature, and in a collapsed condition. A swelling in the left groin was regarded as suspicious, and juice taken from the gland revealed the presence of the *B. pestis.* She died on the 8th April. The child was born apparently quite well, but died five days after birth. *Post-mortem* examination of the infant's body revealed the presence of plague bacilli in the spleen and heart blood.

A sister, aged fourteen, of the above woman, had visited the latter in her home at Duncan street, Valley, about the time of the onset of the latter's illness and previous to her removal to the lying-in hospital. She was attacked with plague on the 8th April, and removed to the isolation hospital on the 9th idem. She died there on the 16th April. The probable source of infection was the same in both cases—viz., the house in Duncan street, which had been overrun with rats. The former woman had picked up a dead rat on the doorstep a few days previous to the onset of her illness, and several dead rats, unfit for examination, were found during disinfecting operations. Fleas were also very numerous in the bedroom. Two healthy guinea-pigs were allowed to run about this room as a trap for rat-fleas for a period of twenty-four hours, but no fleas were taken.

A fatal case, reported after death, a man, aged thirty-five years, employed as a carter in a produce store, occurred at Ipswich early in February.

A fatal case, also reported after death, occurred at Townsville on 17th March. This patient was also employed in a produce store where dead rats were subsequently obtained.

Whether plague manifests itself in atypical clinical forms at the beginning of an epidemic is a question which has a practical bearing in connection with the difficulty not infrequently associated with the recognition of plague appearing for the first time in a new place.

Sporadic cases of benign glandular enlargement have often been recognised during the epidemics above discussed, and bacteriological examination of the "specimens" obtained from these cases have proved conclusively the presence of the *B. pestis.* On the other hand, it has been our experience that in no epidemic of plague reported at Brisbane have we had any practical observation of a large proportion of cases, preceding or following plague epidemics, which could be designated as pestis minor.

Towards the latter end of January, 1907, some thirty-one cases of illness were reported by the Health Officer at Port Douglas. These cases occurred in the Mossman district, a sugar-growing locality some 15 miles from Port Douglas, a new district not previously infected. All the sufferers were cane-cutters, established in camps distant 6 to 7 miles or more from the Mossman Sugar-mill. The epidemic was characterised by slight glandular enlargements in the groins, arm-pits, and neck, raised temperature, slow and low tension pulse, headache, furred tongue, slight congestion of the eyes, and some delirium. The symptoms lasted four or five days, and the patients then recovered. In but few cases were distinct or definite buboes noticed, and there was little glandular tenderness or periadenitis. In seven cases the phenomena were more pronounced, and juice taken from a more or less defined bubo was found to contain plague bacilli. The result of simple microscopic examination of the contents of enlarged glands in the majority of the cases was negative, tube cultures and guinea-pig inoculations not being carried out until towards the close of the epidemic. In three cases, while the bacterioscopic examination was negative, culture and inoculation evidence were positive of *Pestis*.

The patients were treated in hospital at Port Douglas and progressed favourably to convalescence.

Cases of *Pestis minor* continued to be reported up to May, and, in all, some sixty cases came under the notice of the local Health Officer. In only ten of the above cases were bacilli morphologically resembling *B. pestis* found in the material forwarded to the Government Bacteriologist. After due and careful consideration of all the clinical and bacteriological reports and other factors in connection with the cases, it was decided to accept as positive of *Pestis* only the abovementioned ten cases. The Departmental Health Officer (Dr. Chesson) who, in company with Mr. Beardmore, of the Bacteriological Laboratory, visited the Mossman district, Port Douglas, early in May, for the purpose of inquiring into the circumstances of the outbreak, reported: "From the cases now under observation material was taken and cover and culture preparations made. Guinea-pigs were also inoculated. The bacterioscopic, cultural, and inoculation tests were in every instance entirely negative of pestis."

The destruction of rats infesting the northern canefields was a problem demanding the most careful consideration, affecting as it did the financial, social, and political aspects of the sugar-growing industry. After consultation with the directors of the mill upon a practicable and effective method of freeing the canefields of its vermin, it was decided to bait the whole of the plantations with poisoned (phosphorous and arsenic) material and with a preparation of the Danysz virus. Fairly good results were subsequently obtained.

On the 23rd May, a girl, aged twelve years, was reported from Ipswich as a case of bubonic plague. The patient resided with her parents on a farm some 6 miles distant from Ipswich, where her father kept a fruit store. The patient had not visited the latter or the town of Ipswich for a period of six weeks prior to her illness. Both the fruit store at Ipswich and the farm residence were thoroughly searched for rats, but no traces of the latter were found.

The case was a severe one of the usual type with cervical glands. The patient died in the Isolation Hospital on 1st June.

Two fatal cases occurred at Wynnum, a seaside resort distant 12 miles from Brisbane, during the month of June. One case, a boy, aged twelve years, was reported after *post-mortem* examination.

The eighth outbreak of plague in Queensland was marked by 49 cases with 19 deaths, distributed as under:—

Place.	Date.	Cases.	Deaths.	Case Mortality.
				Per cent.
Brisbane	January-June	36	14	39·0
Ipswich	12th February ... ... 23rd May	2	2	100·0
Port Douglas	January-May	10	2	20·0
Townsville	17th March	1	1	100·0
	Total	49	19	38·8

The 19 fatal cases were all among whites, and 6 cases were reported after death.

TYPE AND TYPE MORTALITY.

—	Bubonic.	Septicæmic.	Pneumonic.
Cases	45	4	0
Deaths	15	4	0

Site of Buboes.—Left femoral, 22; right femoral, 7; right inguinal, 3; left inguinal, 2; left axillary, 2; right submaxillary, 1; left cervical, 1; not stated, 11; total, 49.

Age Incidence.

NUMBER OF ATTACKS AND DEATHS UNDER SEX AND AGE GROUPS.

Age Group.	-5.		-10.		-15.		-20.		-25.		-30.		-40.		-45.		-50.		-60.		Over 60.		Total.
Sex.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	
Attacks... ...	1	0	4	1	6	3	8	0	5	1	1	0	6	0	4	1	5	0	1	1	0	1	49
Deaths	1	0	1	0	3	2	1	0	0	1	0	0	4	0	1	0	2	1	0	1	0	1	19

APPENDICES TO PART I.

TABLE I.—Showing Incidence of Plague on Human Beings in the State of Queensland for whole Period 1900 to 1907 (30th June).

Area.	1900.	1901.	1902	1903.	1904.	1905.	1906.	1907.	Total Cases.	Total Deaths.
Brisbane (Metropolitan Area)—										
Attacks	56	36	82	21	30	28	11	36	300	...
Deaths	25	12	26	11	8	15	7	14	...	118
Ipswich—										
Attacks	1	0	0	0	1	8	0	2	12	...
Deaths	0	0	0	0	1	2	0	2	...	5
Maryborough—										
Attacks	0	0	0	0	2	10	0	0	12	...
Deaths	0	0	0	0	1	8	0	0	...	9
Childers—										
Attacks	0	0	0	0	0	1	0	0	1	...
Deaths	0	0	0	0	0	1	0	0	...	1
Bundaberg—										
Attacks	0	0	1	2	0	1	0	0	4	...
Deaths	0	0	1	1	0	1	0	0	...	3
Gladstone—										
Attacks	0	0	1	0	0	0	0	0	1	...
Deaths	0	0	1	0	0	0	0	0	...	1
Rockhampton—										
Attacks	36	0	0	2	0	0	11	0	49	...
Deaths	21	0	0	2	0	0	4	0	...	27
Townsville—										
Attacks	37	0	7	3	0	6	0	1	54	...
Deaths	9	0	5	2	0	6	0	1	...	23
Cairns—										
Attacks	5	0	0	1	2	2	10	0	20	...
Deaths	2	0	0	1	2	0	1	0	...	6
Port Douglas—										
Attacks	0	0	0	0	0	0	0	10	10	...
Deaths	0	0	0	0	0	0	0	2	...	2
Charters Towers—										
Attacks	1	0	0	0	0	0	0	0	1	...
Deaths	0	0	0	0	0	0	0	0	...	0
Totals { Cases	136	36	91	29	35	56	32	49	464	195
Totals { Deaths	57	12	33	17	12	33	12	19	...	...

TABLE II.—Statement Showing the General Mortality Throughout the State for Years 1900-1907.

No.	Place.	Number of Epidemics.	Years.	Total Number of Attacks from January, 1900, to June, 1907.	Total Number of Deaths.	Case Mortality Percentages (Gross).
1.	Brisbane	8	1900 1904 1901 1905 1902 1906 1903 1907	300	118	39·3
2.	Ipswich	4	1900 1904 1905 1907	12	5	41·7
3.	Maryborough	2	1904 1905	12	9	75·0
4.	Bundaberg	3	1902 1903 1905	4	3	75·0
5.	Gladstone	...	1902	1	1	100·0
6.	Childers	...	1905	1	1	100·0
7.	Rockhampton	3	1900 1903 1906	49	27	55·1
8.	Townsville	4	1900 1902 1903 1905 1907	54	23	42·5
9.	Cairns	5	1900 1903 1904 1905 1906	20	6	30·0
10.	Port Douglas	1	1907	10	2	20·0
11.	Charters Towers	...	...	1	...	Nil
	Totals	30	...	464	195	42·0

TABLE III.—SHOWING RACE INCIDENCE, {ATTACKS, DEATHS,} AND CORRECTED DEATH-RATE FOR WHITE RACES, 1900-1907 (WHOLE STATE).

Year.	Whites.	Chinese.	Kanakas.	Aboriginals.	Japanese,	Hindoos.	Number of Attacks Deaths.	Cases—Mortality.	Corrected Death Rate for White Races
								Per Cent.	Per Cent.
1900 ...	126 Attacks 48 Deaths	9 8	1 1	... ...	... ...	... ...	136 57	41·9	38·09
1901 ...	35 Attacks 12 Deaths	1 0	... ...	... ...	... ...	... ...	36 12	33·3	34·2
1902 ...	88 Attacks 30 Deaths	3 3	... ...	... ...	... ...	... ...	91 33	36·2	34·09
1903 ...	25 Attacks 13 Deaths	4 4	... ...	... ...	... ...	... ...	29 17	58·6	52·0
1904 ...	31 Attacks 8 Deaths	2 2	1 1	1 1	... ...	... ...	35 12	34·2	25·8
1905 ...	53 Attacks 30 Deaths	2 2	... ...	1 1	... ...	... ...	56 33	58·9	56·6
1906 ...	30 Attacks 10 Deaths	1 1	... ...	... ...	1 1	... ...	32 12	37·5	33·3
1907 ...	49 Attacks 19 Deaths	...	...	...	...	...	49 19	38·7	38·7

TABLE IV.—SHOWING RELATIVE FREQUENCY OF BUBONIC AND NON-BUBONIC PLAGUE.

YEAR.	TOTAL CASES OBSERVED.	BUBONIC CASES.		NON-BUBONIC CASES.					
				PNEUMONIC (PRIMARY).		SEPTICÆMIC.		TOTAL.	
		Number Observed.	Per Cent. to Total Cases Observed.	Number Observed.	Per Cent. to Total Cases Observed.	Number Observed.	Per Cent. to Total Cases Observed.	Number Observed.	Per Cent. to Total Cases Observed.
1900	136	127	93·3	0	0	9	6·6	9	6·6
1901	36	32	88·8	0	0	4	11·1	4	11·1
1902	91	86	94·4	2	2·1	3	3·2	5	5·4
1903	29	29	100·0	0	0	0	0	0	0
1904	35	31	88·5	0	0	4	11·4	4	11·4
1905	56	41	73·2	3	5·3	12	21·4	15	26 7
1906	32	27	84·3	0	0	5	15·5	5	15 5
1907	49	41	83·6	0	0	8	16·3	8	16·3
GRAND TOTAL ...	464	414	89·2	5	1·0	45	9·6	50	10·7

Type of Plague.	Number of Cases Observed.	Case Mortality per cent. gross.	Correeted Death Rate for Whites per cent. (all types of Plague).
Bubonic (including cases of "Pestis Minor"	414	36·7	39·0
Septicæmic	45	82·0	
Pneumonic	5	100·	

TABLE V.—AGE INCIDENCE SHOWING THE NUMBER OF ATTACKS AND OF DEATHS DISTRIBUTED UNDER AGE GROUPS FOR WHOLE PERIOD, 1900 TO 1907.

Age Groups.	-5.	-10.	-15.	-20.	-25.	-30.	-35.	-40.	-45.	-50.	-55.	-60.	-65.	-70.	Over 70.	Total.
Attacks	16	38	58	49	59	54	38	42	33	30	12	12	8	3	1	453
Deaths	10	21	24	20	21	24	15	17	10	13	9	5	3	2	1	195

Age not noted in 11 cases (attacks).

TABLE VI.—Showing Sex Incidence and Mortality.

—		1900.	1901.	1902.	1903.	1904.	1905.	1906.	1907.	Total.
Cases	Males	114	27	63	22	29	38	25	41	359
	Females	22	9	28	7	6	18	7	8	105
	Total	136	36	91	29	35	56	32	49	464
Deaths	Males	47	11	19	15	11	21	12	14	150
	Females	10	1	14	2	1	12	0	5	45
	Total	57	12	33	17	12	33	12	19	195

METROPOLITAN AREA.

Table VII.—Showing Monthly Returns of Human and Rat Plague for the Years 1900-1907.

Month.	Human Plague.	Rats Examined.	Rats Infected.	Per cent. Examined Infected.
1900.				
January	...	...	...	...
February	...	...	...	...
March	...	13	5	38·46
April	3	4	3	75·00
May	11	1	...	...
June	8	46	15	32·60
July	15	110	21	19·00
August	4	109	24	22·02
September	5	96	15	15·62
October	4	152	7	4·60
November	4	120	...	...
December	2	84	...	...
Totals for year	56	735	90	12·24
1901.				
January	...	44	...	...
February	1	...	...	...
March	7	48	...	...
April	7	169	16	9·47
May	12	163	23	14·10
June	3	121	32	26·45
July	3	144	20	13·89
August	1	136	8	5·88
September	...	245	1	0·40
October	1	332	...	...
November	...	273	1	0·37
December	1	176	...	...
Totals for year	36	1,851	101	5·45
1902.				
January	1	236	3	1·27
February	13	246	6	2·44
March	21	156	7	4·49
April	28	337	53	15·73
May	18	143	16	11·19
June	...	147	12	8·16
July	...	109	8	7·34
August	1	257	1	0·39
September	...	387	...	...
October	...	583	...	...
November	...	527	...	...
December	...	723	...	...
Totals for year	82	3,851	106	2·75

METROPOLITAN AREA—*continued.*

Month.	Human Plague.	Rodents Examined.	Rodents Infected.	Per cent. Examined Infected
1903.				
January	...	1,157	1	0·09
February	5	725	8	1·10
March	4	1,678	30	1·79
April	6	1,226	20	1·63
May	4	1,035	15	1·45
June	...	1,293	6	0·47
July	...	1,111	...	...
August	...	918	...	...
September	2	952	4	0·42
October	...	1,219	...	...
November	...	1,579	...	...
December	...	1,260	...	...
Totals for year	21	14,153	84	0·59
1904.				
January	...	2,220	21	0·95
February	5	1,917	3	0·15
March	...	2,528	25	0·99
April	7	2,971	25	0·84
May	11	2,557	89	3·48
June	1	2,745	150	5·46
July	...	1,468	38	2·59
August	5	1,162	17	1·46
September	1	1,556	4	0·26
October	...	2,157	...	...
November	...	1,456	6	0·41
December	...	1,915	2	0·10
Totals for year	30	24,652	380	1·54
1905.				
January	4	1,572	5	0·32
February	16	2,187	59	2·69
March	2	2,217	48	2·17
April	1	1,561	3	0·19
May	4	1,766	7	0·40
June	1	798	5	0·62
July	...	892	1	0·11
August	...	935	...	...
September	...	1,382	...	...
October	...	1,270	...	...
November	...	1,019	1	0·10
December	...	1,181	...	...
Totals for year	28	16,780	129	0·77
1906.				
January	...	1,031	1	0·09
February	...	1,203	6	0·50
March	2	1,488	6	0·40
April	2	1,312	3	0·24
May	2	1,448	5	0·34
June	2	1,461	6	0·41
July	...	567	2	0·35
August	...	718	8	1·12
September	...	364	...	...
October	...	522	1	0·19
November	2	795	...	...
December	1	1,286	10	0·78
Totals for year	11	12,195	48	0·40
1907.				
January	21	1,249	4	0·32
February	3	1,574	6	0·38
March	3	1,751	1	0·06
April	4	1,494	5	0·33
May	4	1,997	3	0·15
June	1	2,314	3	0·13
	36	10,379	22	0·21

METROPOLITAN AREA.

TABLE VIII.—Showing Weekly Course of the Epidemic and Epizoötic for Years 1900-1907.

Year.	Weeks.	January.				February.				March.					April.				May.				June.					July.				August.				September.					October.					November.				December.				Totals.
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52	
1900	Epidemic ...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	1	1	7	2	1	...	3	1	5	7	1	2	4	...	2	1	1	...	2	3	...	...	...	...	4	...	2	...	...	2	...	1	1	...	...	56
	Epizoötic ...	...	...	...	...	...	...	...	...	...	5	...	...	...	...	...	1	2	...	...	...	...	...	...	2	8	5	2	5	5	6	6	3	5	8	5	4	7	3	1	1	...	5	1	...	...	...	...	...	...	...	...	...	90
1901	Epidemic ...	...	...	...	...	...	...	...	...	1	2	2	1	2	3	1	2	1	1	5	3	...	3	1	...	...	2	2	...	...	1	...	...	1	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	1	...	...	...	36
	Epizoötic ...	..	...	...	...	...	...	...	...	...	...	...	...	...	1	1	6	5	8	5	4	3	6	7	8	10	10	7	3	2	4	2	5	2	...	...	...	...	...	1	...	...	...	...	...	...	...	1	...	...	...	...	...	101
1902	Epidemic ...	..	...	...	1	1	3	2	4	3	11	2	5	3	2	6	7	11	8	5	3	1	3	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	82
	Epizoötic ...	...	..	1	1	1	1	...	1	5	1	2	2	1	10	26	5	7	7	5	4	4	1	4	4	2	2	3	3	...	...	2	1	...	...	...	...	...	...	...	...	...	..	...	...	...	...	...	...	...	...	...	...	106
1903	Epidemic ...	...	...	...	...	...	1	2	1	1	2	1	1	1	1	1	1	2	2	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	21
	Epizoötic ...	...	1	...	...	1	4	1	2	1	3	7	7	16	2	6	4	4	5	5	1	...	6	4	...	...	...	...	...	...	...	...	...	...	...	...	...	1	2	1	...	...	...	...	...	...	...	...	...	..	...	...	...	84
1904	Epidemic ...	..	...	...	...	...	2	3	...	...	...	...	...	...	...	2	2	1	5	2	2	3	1	...	1	...	...	..	...	...	...	...	...	2	3	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	30
	Epizoötic ...	...	1	3	15	2	1	1	...	3	4	2	4	13	3	8	2	5	16	17	14	9	96	24	41	8	21	24	6	3	4	12	1	2	1	3	2	...	...	...	1	...	...	...	...	2	2	2	...	...	2	...	...	380
1905	Epidemic ...	1	..	1	1	4	4	7	1	1	2	...	...	1	...	...	...	...	...	...	2	...	2	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	28
	Epizoötic ...	2	2	1	...	9	12	13	17	24	23	6	3	...	2	1	...	...	...	1	1	3	3	...	1	3	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	129
1906	Epidemic ...	...	...	...	...	...	..	..	...	...	1	...	...	1	...	2	...	...	1	...	1	...	...	1	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	1	...	...	...	1	...	11
	Epizoötic ...	...	...	...	...	1	5	1	...	...	4	2	...	...	2	1	...	...	...	...	...	5	...	5	...	...	1	...	...	1	...	5	2	1	1	...	...	...	...	...	...	...	...	1	...	...	...	...	...	2	1	6	1	48
1907	Epidemic ...	1	1	12	5	3	1	...	1	...	2	1	...	...	1	3	...	...	...	...	1	3	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	36
	Epizoötic ...	1	2	...	1	1	...	1	3	1	1	...	...	...	1	2	2	...	...	1	...	...	2	1	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	22

METROPOLITAN AREA—1900-1907.

TABLE IX.—Showing Monthly Analysis of Rats and Mice Destroyed, Examined, and Infected.

1900.

Month.	Destroyed.		Examined.		Infected.	
	Rats.	Mice.	Rats.	Mice.	Rats.	Mice.
January	No record kept.	Ditto.	...	...	...	...
February			...	...	...	...
March			13	...	5	...
April			4	...	3	...
May			1	...	...	...
June			46	...	15	...
July			110	...	21	...
August			109	...	24	...
September			96	...	15	...
October			152	...	7	...
November			120	...	...	...
December			84	...	...	...
Totals for Year ...	...	...	735	...	90	...

1901.

Month.	Destroyed.		Examined.		Infected.	
	Rats.	Mice.	Rats.	Mice.	Rats.	Mice.
January	No record kept.	Ditto.	44	...	...	...
February			...	...	...	...
March			48	...	...	...
April			169	...	16	...
May			163	...	23	...
June			121	...	32	...
July			144	...	20	...
August			136	...	8	...
September			245	...	1	...
October			332	...	...	...
November			273	...	1	...
December			176	...	...	...
Totals for Year ...	...	...	1,851	...	101	...

1902.

Month.	Destroyed.		Examined.		Infected.	
	Rats.	Mice.	Rats.	Mice.	Rats.	Mice.
January	No record kept.	Ditto.	236	...	3	...
February			246	...	6	...
March			156	...	7	...
April			337	...	53	...
May			143	...	16	...
June			147	...	12	...
July	255	...	109	...	8	...
August	704	...	257	..	1	...
September	741	...	387	...	...	...
October	952	...	583	...	...	...
November	797	...	527	...	...	...
December	1,222	..	723	...	...	...
Totals for Year ...	4,671	...	3,851	...	106	..

1903.

Month.	Destroyed.		Examined.		Infected.	
	Rats.	Mice.	Rats.	Mice.	Rats.	Mice.
January	1,662	...	1,157	...	1	...
February	1,428	30	718	7	7	1
March	3,863	10	1,674	4	29	1
April	2,251	...	1,226	...	20	...
May	1,305	97	1,035	...	15	...
June	1,753	30	1,293	...	6	...
July	1,428	...	1,111	...	...	...
August	1,340	...	918	...	...	...
September	4,095	...	952	...	4	...
October	1,261	...	1,219	...	...	...
November	1,860	17	1,579	...	...	...
December	1,620	...	1,260	...	...	...
Totals for Year ...	23,866	184	14,142	11	82	2

1904.

Month.	Destroyed.		Examined.		Infected.	
	Rats.	Mice.	Rats.	Mice.	Rats.	Mice.
January	2,397	...	2,220	...	21	...
February	3,000	276	1,917	...	3	...
March	7,114	7,448	2,487	41	25	...
April	2,879	3,662	1,784	1,187	25	...
May	2,850	1,999	1,604	953	86	3
June	3,278	2,217	1,684	1,061	150	...
July	1,597	1,260	1,141	327	38	...
August	1,031	317	873	289	17	...
September	1,679	286	1,363	193	4	...
October	3,172	354	1,786	371	...	...
November	1,941	322	1,204	252	6	...
December	3,109	378	1,626	289	2	...
Totals for Year ...	34,047	18,519	19,689	4,963	377	3

1905.

Month.	Destroyed.		Examined.		Infected.	
	Rats.	Mice.	Rats.	Mice.	Rats.	Mice.
January	2,734	522	1,242	330	5	...
February	3,241	816	1,669	518	58	1
March	3,730	660	1,703	514	48	...
April	2,453	362	1,248	313	3	...
May	2,635	402	1,424	342	7	...
June	1,379	448	549	249	5	...
July	1,380	268	697	195	1	...
August	1,473	325	753	182	...	...
September	1,783	469	1,102	280	...	...
October	1,871	359	1,035	235	...	...
November	1,497	388	845	174	1	...
December	1,811	369	1,016	165	...	...
Totals for Year ..	25,987	5,388	13,283	3,497	128	1

1906.

Month.	Destroyed.		Examined.		Infected.	
	Rats.	Mice.	Rats.	Mice.	Rats.	Mice.
January	1,608	242	895	136	1	...
February	1,794	188	1,105	98	6	...
March	2,251	389	1,248	240	6	...
April	1,865	385	1,142	170	3	...
May	2,323	271	1,311	137	5	..
June	2,063	277	1,317	144	6	...
July	600	108	467	100	2	...
August	908	108	634	84	8	...
September	535	45	332	32	...	...
October	708	98	447	75	1	...
November	956	83	729	66	...	...
December	1,720	122	1,220	66	10	...
Totals for Year ...	17,331	2,316	10,847	1,348	48	...

1907.

Month.	Destroyed.		Examined.		Infected.	
	Rats.	Mice.	Rats.	Mice.	Rats.	Mice.
January	1,773	104	1,164	85	4	...
February	2,306	243	1,436	138	6	...
March	2,633	219	1,593	158	1	...
April	1,808	204	1,322	172	5	...
May	2,289	228	1,794	203	3	...
June	2,626	375	1,945	369	3	...
July						
August						
September						
October						
November						
December						
Totals for Year ...	13,435	1,373	9,254	1,125	22	...

METROPOLITAN AREA—BRISBANE, 1904.

TABLE X.—Showing Weekly Analysis of Rats and Mice Examined, Destroyed, and Infected, 1904.

For Week ending—	Rats Examined.	Mice Examined.	Rats not Examined.	Mice not Examined.	Rats and Mice Destroyed.	Rats Infected.	Per Cent.	Mice Infected.	Per Cent.
9 January	519	...	15	...	534	1	0·192	...	...
16 ,,	498	...	24	...	522	1	0·201	...	...
23 ,,	513	...	21	...	534	6	1·170	...	...
30 ,,	690	...	17	...	707	13	1·884	...	...
6 February	506	...	42	...	548	...	...	...	...
13 ,,	471	...	34	...	505	1	0·210	...	...
20 ,,	395	...	27	...	422	1	0·253	...	...
27 ,,	463	...	462	8	933	...	...	...	...
5 March	462	...	1,251	560	2 273	4	0·866	...	...
12 ,,	495	9	1,477	1,593	3 574	3	0·606	...	...
19 ,,	436	38	1,020	1,777	3,271	5	1·146	...	...
26 ,,	524	196	1,202	1,975	3 897	4	0·763	..	...
2 April	368	186	916	1,585	3.055	10	2·717	...	...
9 ,,	362	247	401	482	1,492	4	1·105	...	...
16 ,,	413	249	241	684	1,587	7	1·697	...	...
23 ,,	446	299	231	739	1,715	3	0·673	...	...
30 ,,	508	345	140	417	1.410	11	2·165	...	...
7 May	443	237	215	221	1.116	15	3·386	...	...
14 ,,	391	222	345	331	1 289	12	3·069	...	...
21 ,,	355	246	263	328	1,192	15	4·225	2	0·812
28 ,,	327	121	224	224	896	27	8·257	...	...
4 June	444	163	289	244	1.140	80	18·018	1	0·613
11 ,,	458	129	297	250	1,134	28	6·111	...	...
18 ,,	583	221	277	277	1,358	36	6·175	...	...
25 ,,	380	208	251	279	1,118	9	2·368	...	...
30 ,,	209	163	178	352	902	14	6·699	..	...
9 July	380	100	150	523	1.153	27	7·105	...	...
16 ,,	436	62	188	463	1,149	4	0 917	...	...
23 ,,	181	79	20	...	280	2	1·105	...	...
30 ,,	190	33	52	...	275	5	2·632	...	...
6 August	236	30	15	...	281	12	5·085	...	...
13 ,,	205	69	32	...	306	2	0·976	...	...
20 ,,	149	98	51	34	332	...	...	...	...
27 ,,	180	42	31	...	253	1	0·555	...	...
3 September	223	76	47	2	348	3	1·346	...	...
10 ,,	233	35	68	..	336	2	0·858	...	...
17 ,,	331	37	71	9	448	..	...	...	...
24 ,,	355	28	125	4	512	...	...	...	...
1 October	394	145	78	9	626	1	0·279	...	...
8 ,,	454	141	240	1	836	...	...	...	...
15 ,,	300	42	98	10	450	...	...	...	...
22 ,,	536	24	647	11	1,218	...	...	...	...
29 ,,	361	96	310	1	768	...	...	...	...
5 November	296	36	170	5	507	...	...	...	...
12 ,,	309	58	168	28	563	3	0·971	...	...
19 ,,	163	62	143	12	380	2	1·233	...	...
26 ,,	304	57	188	9	558	1	0·329	...	...
3 December	469	90	236	35	830	...	...	...	...
10 ,,	554	40	255	22	871	...	...	...	...
17 ,,	368	91	657	28	1,144	2	0·598	...	...
24 ,,	229	39	299	22	589	...	...	...	...
31 ,,	194	74	159	2	429	...	...	...	...
	19,689	4,963	14,358	13,556	52,566	377	1·915	3	0·060

Cost of rat destruction	£3,019 19s. 10d.

Species.

Mus. decumanus	309
Mus. Alexandrinus	32
Mus. rattus	31
Mus musculus, and not given	8
Total	380

METROPOLITAN AREA—BRISBANE, 1905.

TABLE XI.—Showing Weekly Analysis of Rats and Mice Destroyed, Examined, and Infected.

For Week Ending—	Rats Examined.	Mice Examined.	Rats not Examined.	Mice not Examined	Rats and Mice Destroyed.	Rats Infected.	Per Cent.	Mice Infected.	Per Cent.
7 January	260	79	390	52	781	2	0·769	...	...
14 ,, ...	306	119	352	109	886	2	0·654	...	...
21 ,, ...	315	33	375	17	740	1	0·314	...	...
28 ,, ...	271	87	311	14	683	...	...	...	...
4 February	364	100	316	3	783	8	2·198	1	1·000
11 ,, ...	410	126	391	117	1,044	12	2·927	...	...
18 ,, ...	398	155	336	98	987	13	3·267	...	...
25 ,, ...	459	122	472	63	1,116	17	3·704	...	...
4 March	561	137	546	57	1,301	24	4·278	...	...
11 ,, ...	398	94	462	13	967	23	5·779	...	...
18 ,, ...	268	72	443	23	806	6	2·239	...	...
25 ,, ...	352	173	431	50	1,006	3	0·852	...	...
1 April	379	98	339	21	837	...	...	...	...
8 ,, ...	245	53	250	11	559	2	0·816	...	...
15 ,, ...	297	90	272	4	663	1	0·337	...	...
22 ,, ...	334	81	417	17	849	...	...	...	...
29 ,, ...	245	56	186	14	501	...	...	...	...
6 May	546	66	314	13	939	...	...	...	...
13 ,, ...	215	58	253	27	553	1	0·465	...	...
20 ,, ...	232	56	102	17	407	1	0·431	...	...
27 ,, ...	217	130	367	3	717	3	1·382	...	...
3 June	304	63	296	23	686	3	0·987	...	...
10 ,, ...	107	58	125	33	323	...	..	...	...
17 ,, ...	135	53	150	52	390	1	0·741	...	...
24 ,, ...	128	50	303	39	520	3	1·562	...	...
30 ,, ...	89	57	138	54	338	...	...	...	...
8 July	161	46	153	18	378	1	0·606	...	...
15 ,, ...	154	35	166	28	383	...	...	...	...
22 ,, ...	135	36	115	14	300	...	...	...	...
29 ,, ...	224	68	217	11	520	...	...	...	...
5 August	152	73	204	39	468	...	...	...	...
12 ,, ...	114	23	147	24	308	..	...	...	...
19 ,, ...	132	34	163	39	368	...	...	...	...
26 ,, ...	257	36	156	25	474	...	...	...	...
2 September	214	51	126	32	423	...	...	...	...
9 ,, ...	255	34	183	48	520	...	...	...	...
16 ,, ...	217	43	126	23	409	...	...	...	...
23 ,, ...	252	126	172	56	606	...	...	...	..
30 ,, ...	285	52	156	48	541	...	...	...	...
7 October	240	51	172	15	478	...	...	...	...
14 ,, ...	354	81	194	26	655	...	...	...	...
21 ,, ...	213	47	199	46	505	...	...	...	...
28 ,, ...	169	45	199	21	434	...	...	...	...
4 November	181	39	164	37	421	...	...	...	...
11 ,, ...	213	42	112	83	450	...	...	...	...
18 ,, ...	192	40	175	50	457	...	..	...	...
25 ,, ...	203	35	170	48	456	...	...	...	...
2 December	185	42	144	21	392	1	0·541	...	...
9 ,, ...	388	69	138	48	643	...	...	..	...
16 ,, ...	235	29	175	68	507	...	...	...	..
23 ,, ...	190	30	226	61	507	...	...	...	...
30 ,, ...	133	24	210	23	390	...	..	...	...
	13,283	3,497	12,699	1,896	31,375	128	0·887	1	0·029

Cost of rat destruction ... ... £1,932 6s. 7d.

Species.

Mus decumanus	87
Mus Alexandrinus	1
Mus rattus	40
Mus musculus	1
	129

METROPOLITAN AREA—BRISBANE, 1906.

TABLE XII.—Showing Weekly Analysis of Rats and Mice Destroyed, Examined, and Infected.

For Week Ending—	Rats Examined.	Mice Examined.	Rats not Examined.	Mice not Examined.	Rats and Mice Destroyed.	Rats Infected.	Per Cent.	Mice Infected.	Per Cent.
6 January	163	26	164	19	372	...	...	...	...
13 ,,	222	41	216	22	501	...	...	...	...
20 ,,	172	32	163	28	395	...	...	...	...
27 ,,	240	20	126	23	409	...	...	...	...
3 February	176	24	110	30	340	1	0·57	...	...
10 ,,	308	23	199	20	550	5	1·62	...	...
17 ,,	322	28	138	17	505	1	0·31	...	...
24 ,,	283	24	188	14	509	...	...	...	...
3 March	235	45	196	32	508	...	..	...	...
10 ,,	217	56	196	36	505	4	1·84	...	...
17 ,,	251	48	274	43	616	2	0·80	..	...
24 ,,	324	44	238	39	645	...	...	...	...
31 ,,	335	63	197	22	617	...	...	...	...
7 April	315	66	178	63	622	2	0·63	...	...
14 ,,	186	26	231	64	507	1	0·54	...	...
21 ,,	274	34	149	33	490	...	...	...	...
28 ,,	316	34	133	45	528	...	...	...	...
5 May	217	49	196	55	517	5	2·30	...	...
12 ,,	326	31	190	28	575	...	...	...	...
19 ,,	324	23	193	25	565	...	...	...	...
26 ,,	285	33	359	14	691	...	...	...	...
2 June	282	27	212	39	560	...	..	...	...
9 ,,	420	49	223	36	728	5	1·19	...	...
16 ,,	286	29	137	39	491	...	...	...	...
23 ,,	339	22	149	15	525	...	...	...	...
30 ,,	200	28	131	26	385	1	0·50	...	...
7 July	53	21	27	4	105	...	...	...	...
14 ,,	100	37	31	0	168	...	...	...	...
21 ,,	174	20	35	3	232	1	0·57	...	...
28 ,,	118	18	37	1	174	...	...	...	..
4 August	88	11	49	3	151	5	5·68	...	...
11 ,,	63	10	41	1	115	2	3·17	...	...
18 ,,	201	20	87	15	323	1	0·50	...	...
25 ,,	166	28	55	5	254	1	0·60	...	...
1 September	157	20	51	0	228	...	...	...	...
8 ,,	98	11	54	8	171	...	...	...	...
15 ,,	73	2	53	3	131	...	...	...	...
22 ,,	100	5	44	2	151	...	...	...	...
29 ,,	42	12	42	0	96	...	...	...	...
4 October	113	7	45	13	178	...	...	...	...
13 ,,	76	19	70	0	165	...	...	...	...
20 ,,	74	23	51	6	154	...	...	...	...
27 ,,	144	11	77	0	232	1	0·75	...	...
3 November	75	17	36	4	132	...	...	...	...
10 ,,	111	23	39	5	178	...	...	...	...
17 ,,	154	8	43	0	205	...	...	...	...
24 ,,	104	17	20	3	144	...	...	...	...
1 December	395	32	133	15	575	...	...	...	...
8 ,,	238	8	64	3	313	2	0·84	...	...
15 ,,	236	5	83	0	324	1	0·42	...	...
22 ,,	357	13	113	1	484	4	1·12	...	...
31 ,,	319	25	218	46	608	3	0·90	...	...
	10,847	1,348	6,484	968	19,647	48	0·44	...	...

Cost of rat destruction ... ... £1,069 10s. 7d.

Species.

Mus decumanus	16
Mus Alexandrinus	1
Mus rattus	31
	48

METROPOLITAN AREA—BRISBANE, 1907.

TABLE XIII.—Showing Weekly Analysis of Rats and Mice Examined, Destroyed, and Infected.

For Week ending—	Rats Examined.	Mice Examined.	Rats not Examined.	Mice not Examined.	Rats and Mice Destroyed.	Rats Infected.	Per Cent.	Mice Infected.	Per Cent.
5 January	145	8	117	0	270	1	0·69	...	...
12 ,, ...	233	14	158	1	406	1	0·43	...	...
19 ,, ...	274	24	122	4	424	1	0·36	...	...
26 ,, ...	354	25	102	3	484	...	...	...	...
2 February	298	25	184	15	522	1	0·34	...	...
9 ,, ...	421	42	214	36	713	1	0·23	...	...
16 ,, ...	363	30	269	44	706	1	0·28	...	...
23 ,, ...	284	29	221	12	546	3	1·05	...	...
2 March	281	34	131	12	458	1	0·35	...	...
9 ,, ...	414	18	214	11	657	1	0·24	...	...
16 ,, ...	405	39	135	12	591	...	..	...	...
23 ,, ...	335	52	223	30	640	...	...	...	...
30 ,, ...	386	41	429	5	861	...	...	...	...
6 April	256	44	102	17	419	...	..	...	...
13 ,, ...	263	41	104	0	408	2	0·76	...	...
20 ,, ...	315	34	115	9	473	2	0·64	...	...
27 ,, ...	327	41	95	4	467	1	0·30	...	...
4 May	628	35	138	1	802	...	...	...	...
11 ,, ...	388	47	122	5	562	1	0·25	...	...
18 ,, ...	380	44	92	10	526	...	...	...	...
25 ,, ...	291	35	105	0	431	...	...	...	...
1 June	313	59	113	11	496	2	0·64	...	...
8 ,, ...	396	43	63	1	503	1	0·25	...	...
15 ,, ...	766	205	406	1	1,378	...	...	...	...
22 ,, ...	422	54	116	2	594	1	0·23	...	...
30 ,, ...	316	62	91	2	471	1	0·31	...	..
Total	9,254	1,125	4,181	248	14,808	22	0·23	...	...

Total cost of rat destruction	£903 15s. 2d.

Species

Mus. decumanus	13
Mus. Alexandrinus...	2
Mus. rattus	7
Total	22

PART II.

B.—THE INCIDENCE OF PLAGUE.

I.—GEOGRAPHICAL FEATURES.

The State of Queensland comprises the north-eastern portion of the continent of Australia. It extends over 18 degrees of latitude from the 29th parallel, which divides it from New South Wales, to within 11 degrees of the Equator. The area of Queensland is 668,497 square miles, inhabited by little over half a million of people, the estimated population on the 30th January, 1905, being 526,369 persons.

Unlike the southern States of New South Wales and Victoria, whose commerce passes almost exclusively through one or two ports only, Queensland's coast line of 2,250 miles presents a number of harbours, which have become the sites of important towns—Brisbane (the capital of the State and most southerly), Maryborough, Bundaberg, Gladstone, Rockhampton, Mackay, Bowen, Townsville, Cairns, Cooktown, and, at the extreme point, Thursday Island.

Queensland has a variety of climate, from the even temperature and comparatively high humidity of the coast to the extremes of summer heat and winter cold in the far North-west.

The State is naturally divided into coastal and inland districts by a range of hills which run nearly parallel to, and not far from, the coast; separating the eastern from the western waters, and supplying the source of numerous rivers which flow eastward to the sea.

The heat of the summer months is made the more disagreeable from the moisture, as the rains of Queensland, unlike the more southern States, prevail in the late summer months. The City of Brisbane (latitude 27½ degrees south, longitude 153 degrees east) is situated on the river Brisbane, about 20 miles from its mouth. The city was founded in 1825, and covers an area of 5½ square miles. The population on the 31st March, 1901, was 119,428 persons, including the suburban districts within 10 miles of the General Post Office. The mean temperature of Brisbane for the year is 68·4 degrees, the relative humidity 68 per cent. (sat. = 100), the mean annual rainfall 48·55 inches.

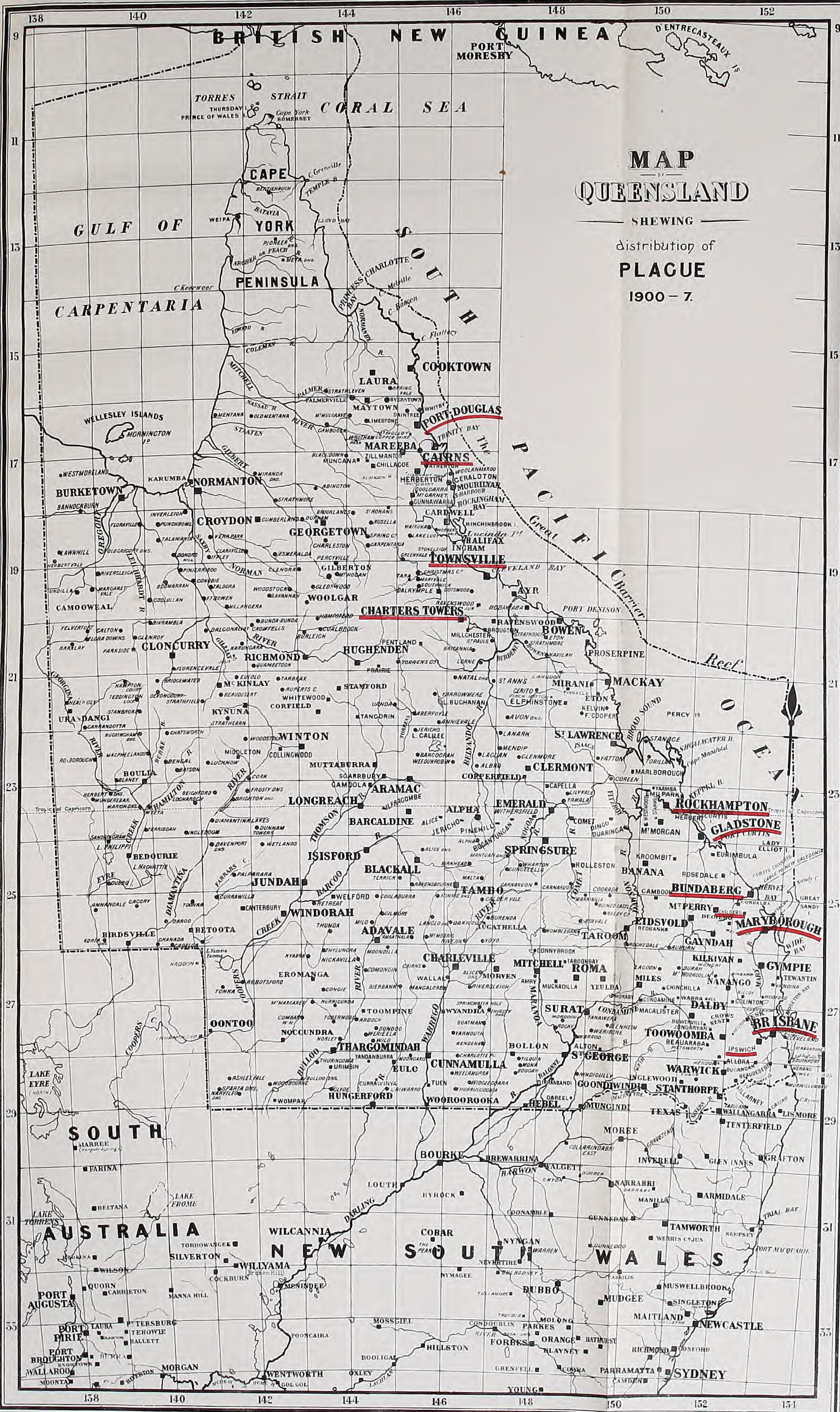
II.—SANITARY ADMINISTRATION.

Prior to "*The Health Act of* 1900," there was a very deficient sanitary organisation. The then existing Central Board of Health was without legal power or a fitting administrative head, the responsibility of plague administration being at first left with the local authorities.

An Order in Council, of 4th April, 1900, provided for the establishment of "Joint Boards for the Prevention of Infectious Diseases," and under this lengthy and somewhat incongruous title any two or more local authorities were empowered, under "*The Health Act of* 1884," to act together for the purpose of the provisions of the said Act relating to the prevention of epidemic diseases.

Some twenty local authorities, within a radius of 12 miles of Brisbane, were combined to form "The Metropolitan Joint Board." This Board, in addition to all other powers vested in it, was empowered to define the limits of any locality which was, or was likely to be, subjected to a visitation of epidemic or infectious diseases; to provide and manage hospitals for the treatment of persons affected with such disease; to arrange for house-to-house visitation, medical aid and accommodation, and the promotion of cleansing and disinfection. It could also take such action generally as, in its opinion, was necessary for ensuring the suppression of such disease. The board was subsidised by the Government to the extent of £1 for £1 on the amounts raised by precepts levied on the local governing bodies comprising the Joint Board.

The outbreak of plague in Brisbane in 1900, and the scare, alarm, and excitement of the populace, even at a name whose association conjured up fearful visions of the "Black Death" of the Middle Ages, led to an urgent demand for sanitary reform, and the passing by the Legislature of "*The Health Act of* 1900."



MAP OF QUEENSLAND

SHEWING distribution of

PLAGUE

1900 – 7.

BY AUTHORITY: G. A. VAUGHAN, GOVT. PRINTER, BRISBANE.

"*The Health Act of* 1900" consolidated and amended prior health legislation, and provided for the appointment of a Commissioner of Public Health (a medical practitioner and expert in sanitary science), who, under the Minister (the Hon. the Home Secretary), was charged with the due and effectual administration of the Act. Enlarged and decided powers were conferred upon the Commissioner, whose "Orders" and "Regulations" were subject to approval by the Minister (or Governor-in-Council), with subsequent appeal, if wished, to Parliament itself.

On the 1st January, 1901, I took up my duties as Commissioner of Public Health.

The organisation called into existence by "*The Health Act of* 1900" was speedily developed. In accordance with the provisions of the Act, a new Central Board of Health was formed, consisting of five members, three being medical practitioners, one member having experience of local government, and one a representative of commerce and trade.

The duties of the new Central Board of Health were purely of an advisory character, without administrative or executive powers: the powers, duties, and liabilities previously vested in or imposed upon the old Central Board being now imposed upon and executed by the Commissioner. The Governor-in-Council also appointed such health officers and sanitary inspectors, together with the necessary clerical staff, as the organisation required.

Regulations for the notification of cases of plague or suspected plague; isolation of the patient, premises, or vessel; removal of patients to hospital; detention of suspected persons; destruction of buildings and things; disinfection and cleansing of premises; curative treatment of patients; control and direction of vessels from infected ports; inoculation of persons as a preventive against disease; destruction and examination of rats; and the establishment of special hospitals, &c., were issued at an early date by the Commissioner, and enforced by the various Joint Boards throughout the State.

Memoranda and circular letters were sent to all medical officers of health, health officers, medical practitioners, &c., detailing the symptoms and diagnostic features of plague, together with directions for obtaining and forwarding for bacteriological examination material from suspected cases. Handbills were also issued by the Department of Public Health for the guidance of the general public as to the manner of spread and the danger to be anticipated with regard to rats.

The mistake, however, of multiplying authorities for the purpose of sanitary administration soon became apparent in the deficient and perfunctory administration of the "Joint Boards." However right in principle as a method of sanitary administration it was not successful in practice; the general mode of procedure adopted by the Joint Boards utterly failing to cope with any emergent circumstances.

In May, 1902, the Commissioner of Health made an "Order" under section 19 of "*The Health Act of* 1900," defining an "Infected Area" of the City of Brisbane, and requesting its cleansing by the Municipal Council and the Metropolitan Joint Board. The "Order" directed the local authority to do its duty in the matter within a time limited by this Order. A gang of fifty men was employed for a period of two months, during which time some 3,000 loads of filth and rubbish were removed from the area, and either jettisoned at sea or destroyed by fire. Immediately following this special cleansing, a notable decrease in the number of cases of plague was observed. From 28 cases in April, and 18 in May, 1902, no further case (with the exception of one sporadic case in August) occurred till the 8th February, 1903—a quiescent period of nearly nine months.

The overlapping of sanitary jurisdiction continued, however, to exist, and it soon became evident that the work of the Commissioner in plague matters would be enhanced in efficiency and diminished in friction were the powers concentrated and consolidated in the one Central Health Authority.

On 19th September, 1902, the Government decided to rescind the Order-in-Council of April, 1900, and the Metropolitan Joint Board and other Boards constituted under similar orders throughout the State were abolished. The administration of the Plague Regulations was transferred from the control of these local governing bodies to the Commissioner of Public Health. Recognising that plague was a disease affecting the whole State, the Government decided to undertake the whole cost of the work of administration and prevention, rather than leave it to the less effective means at the disposal of the local authorities. In all the more important towns, and at all the ports of the State, a Health Officer, acting as the deputy of the Commissioner, was appointed for the proper execution of the Regulations.

The following is a brief scheme of action adopted:—

1. Notification of all cases of plague, or suspicious cases of plague, to the Commissioner and the local Health Officer.
2. Investigation of the case by the local Health Officer.
3. Bacteriological examination of "specimens" by the Health Officer and the Government Bacteriologist.
4. Isolation of patient in the Plague Hospital.
5. Supervision of "original" contacts during the incubation period (segregation now abandoned except in cases of pneumonic plague, or other cases with pulmonary symptoms).
6. Disinfection of infected premises, clothing, bedding, &c.
7. Cleansing and disinfection of infected areas.
8. Destruction of rats.
9. Bacteriological examination (daily) of rats.
10. Gratuitous supplies of curative serum to Health Officers and staffs of general hospitals.
11. Protective inoculation.
12. Inspection of areas for detection of nuisances.
13. Dissemination of literature on plague among Medical Officers of Health, medical practitioners, local authorities, and the public generally.
14. Fumigation of coastal vessels while empty.
15. Shipping regulations relating to the berthing of vessels from infected ports. Gratuitous supplies of rat poison and disinfectants to the local authorities.

Acting in the spirit and letter of the Venice and Paris Conventions, to which the State was a signatory, the Government of Queensland has always given the fullest information to the other States of the Commonwealth and to the authorities abroad, and made public the fact as to the existence of plague in man or rats within the State. The routine method of notification and report instituted reduced the chances of diagnostic error to a minimum. The procedure adopted in Brisbane was as follows:—

Immediately on receipt of information of suspicious illness, the Health Officer to the Department was despatched to visit the case, and examine it clinically, and in consultation with the medical practitioner in attendance if the latter so desired. "Specimens" from the affected gland, blood, or sputum, as the circumstances indicated, were taken; cover preparations of part of this material were made, and the remainder forwarded to the Government Bacteriologist, who also examined independently a smear preparation and inoculated a serum tube and a guinea-pig. The results of the clinical and immediate bacterioscopic examinations were embodied in a report to the Commissioner, to which the subsequent reports on culture and animal inoculation were appended. A diagnosis of plague having been arrived at, the patient was taken to the Plague Hospital and arrangements made for the necessary disinfection and cleansing of the room from which the patient was removed, together with an active search for rats on the dwelling-house and the place of business.

All nuisances within the meaning of the Health Act, or sanitary defects, observed on the premises by the staff inspector were reported to the local authority concerned.

In March of 1903, a special crusade was directed against the rats of Brisbane, which had at that time increased in large numbers. A departmental gang of 120 men was engaged to carry out the work of destruction within the Metropolitan Area. The work extended over a period of six weeks; a permanent gang of 18 men being subsequently employed during the rest of the year. In all, some 24,050 rats were collected; 14,153 rats were examined bacteriologically, of which latter 84 were found to be plague-infected. The actual number of rats destroyed by the gangs was out of all proportion to the number collected for examination, and it was difficult to estimate the effect on the rat population which had been produced. After taking poisoned baits, rats retired to their holes or hiding places to die, and it is obvious that the actual number of rats killed by householders would, if added to the total actually gathered by the gang, materially increase the grand total.

Special cleansing and rat-destruction operations were also carried out at Townsville and other places with excellent results. Specimens of rats—infected and healthy—were collected from Brisbane, Townsville, Rockhampton, Bundaberg, Gladstone, Ipswich, &c., and referred to the Director of the Queensland Museum (Mr. C. de Vis) for purposes of identification and classification.

Several hundred fleas captured on the rats—infected and healthy—were also referred to the Government Entomologist (Mr. Henry Tryon) for report as to their specific identity.

When plague again made its appearance in Brisbane, in January, 1904, the majority of the early cases were associated with the handling and distribution of produce. An "Infected Area," including the produce and grain stores of the city, was declared, and a systematic inspection and cleansing of this area carried out.

In view of the probability of contamination of old and used produce bags by infected rats, some 61,600 bags were disinfected in the Reck apparatus at the Infectious Disease Hospital. Statutory notices were served on the local authority to compel owners of produce and grain stores to provide rat-impervious floors, and to take such other measures as would effectually prevent the access or harbourage of rats on these premises. In several cases new produce stores were erected, old buildings pulled down or abandoned, while structural defects were remedied in the majority of instances and the premises made as rat-proof as possible.

During the months of February-July, a sum of £406 was spent by the Government in capitation fees; 6d. apiece being offered for rats and 3d. for mice.

To the Department's efforts the Brisbane Municipal Council lent ready co-operation, but the indifference and apathy of a certain section of the public, and the active opposition on the part of certain prominent members of the local governing bodies justified the drastic measures the Commissioner found it necessary to take in the enforcement of his cleansing orders. In August, 1904, a recrudescence of plague occurred in South Brisbane and threatened to assume serious proportions. An "emergency," under section 20 of the Health Act, was declared by the Commissioner. Under that section of the statute the Commissioner may exercise, undertake, and perform any or all of the powers and duties vested in or imposed upon a local authority by the above Act or its Regulations; all costs and expenses incurred under and in pursuance of the provisions of this section being paid by the defaulting local authority. While it is the legitimate duty of the Department of Public Health to initiate and undertake all reasonable measures of plague prevention, it is the duty of a local authority under the Health Act to eliminate all conditions affecting, or threatening to affect, injuriously the public health within its area; to require abatement of nuisances existing therein; to remove filth and garbage from the neighbourhood or premises; and to effect improvements in properties specially liable to rat-infestation.

A gang of fifty men was employed in cleansing and rat-destruction operations in the South Brisbane Area for a period of one month, the departmental gang being increased by the addition of thirty men employed at the expense of the local authority.

In all, some 992 loads of rubbish were removed or burned; a flock mill (where two cases of plague had occurred) with its contents destroyed by fire; dilapidated premises condemned or renovated; the breeding-places of rats broken up, and the disease-carrying vermin reduced to a minimum.

As on former occasions, a period of quiescence followed these special cleansing operations.

On the 14th June, 1905, an official inquiry was held by the Commissioner at the Municipal Chambers, Maryborough, into the circumstances of an outbreak of plague in that city. The details of the epidemic are given elsewhere in this Report.

Most important of the measures taken for the prevention and suppression of the outbreaks of plague in the State was the systematic destruction of rodents. A permanent gang, attached to the Department, of from ten to twenty men—increased to 100 as occasion required—has been employed every day for the last four years in laying poisoned baits, trapping, digging out burrows, and destroying the habitual haunts of the rodents within the Metropolitan Area. Rat-catchers also have been permanently employed by the Government at every port and important town throughout the State.

A large percentage of all the rats collected in the Metropolitan Area were examined daily at the Bacteriological Institute, and a daily report thereon submitted.

Capitation fees of 6d. each for rats, and 3d. for mice, have been offered from time to time at various places in the State where plague has made its appearance.

A great variety of rat poison has been tried at periods with varying success; phosphorous with a basis of glucose, syrup, and flour giving the best results. The preparation was spread on thin small squares of bread, and made attractive with the addition of the oils of rhodium or aniseed.

Cultures of Danysz rat virus were obtained and used during the year 1905, but without any useful results being attained. In 1906 and again in 1907 fresh supplies of the Danysz bacillus were secured, as also cultures of the rat virus of Laroche and the "Azoa" rat virus. On these occasions, as mentioned elsewhere, the results obtained were fairly satisfactory.

In connection with rat destruction, it must be remembered that the spread of the disease by rats was a fact which only received general recognition subsequent to the enactment of "*The Health Act of* 1900." While it is the duty of the Health Department to act as a thinking, collating, and initiating body, it is also the duty of the State Health Authority to compel, where necessary, the remedying of sanitary evils produced by local inefficiency and apathy.

In March of 1906, regulations for the better prevention of plague by rats were made by me, and approved by the Governor in Council. It is, however, necessary to make certain amendment of "*The Health Act of* 1900," to meet the newly recognised danger. Until such legislation can be passed empowering local authorities to prosecute occupiers or owners of premises for allowing conditions favourable to infestation by rats to occur and continue upon their premises, it is imperative that the Health Department should still continue to shoulder the responsibility entrusted to it.

A Metropolitan Building Act, or its provision included in an amending Health Statute, is urgently needed.

With regard to quarantine efforts at prophylaxis, it may be said that the Plague Regulations have not come up to what was expected of them by the delegates to the Australian Intercolonial Plague Conference of 1900. Next to the beneficial efforts of such Regulations, the extent to which they may hamper the trade and commerce of the port must be considered. The rigorous and one-sided measures adopted by one of the States of the Commonwealth, whilst not ensuring the protection against plague anticipated, did, however, inflict heavy damage on interstate commercial relations. The proposed federal legislation on quarantine should secure that uniformity of action and certainty of procedure in the administration of the plague (shipping) regulations of the various States which, up to the present, have been notoriously absent.

The history of plague epidemics in the Old World repeats itself in the New, and will continue to do so as long as human nature remains what it is. From terror amounting to almost panic and feverish activity on the part of local authorities and individuals when the dread disease first makes its appearance among a hitherto uninfected community, there arises sooner or later an apathy, an indifference, an inertia produced by a succession of epidemics with moderate death rates. This policy of *laissez-faire* on the part of the local governing bodies seems to be in direct proportion to the activity of the responsible State Health Authority. While the Government with its great monetary resources should render the local authorities reasonable assistance in the efforts of the latter bodies to prevent cases of plague spreading within their areas, there is a line to be drawn between encouraging local bodies or individuals to be sanitary and encouraging them to be helpless. Beyond constant complaint by householders and occupiers of premises of rats on their premises, no systematic assistance of any value was given to the Health Department to cope with the evil. The tendency to deny the existence of plague on its first appearance in any particular place is a world-known fact. The opinion that the disease was not true plague, because it had not manifested the epidemicity characteristic of the disease in India and the East, has been freely expressed in Brisbane and other places in this State, and this opinion has not been confined to the ignorant, notwithstanding the fact that the clinical features, the pathological appearances, and the bacteriological investigations stamped the disease undoubtedly as plague.

A Report on Plague in Brisbane, 1901-1902, was published by Dr. Halford, in July, 1902; and Dr. Baxter-Tyrie has supplemented this with an unofficial account of an outbreak of plague in Queensland during 1904, published in "The Journal of Hygiene," July, 1905. An attempt has been made to collate the results of 1900, but the executive work and deficient organisation during the first year of plague doubtless militated against the keeping of any reliable detailed accounts.

All the patients in Brisbane were isolated and treated at the "Colmslie" Plague Hospital, situated in extensive grounds some three miles distant by road from the city. A horse ambulance conveyed the patients thither, a nurse with experience of plague accompanying each case.

III.—THE INCIDENCE OF PLAGUE.

The circumstances which influence individual liability to plague may be conveniently considered under the following headings:—1. Sex Incidence; 2. Age Incidence; 3. Race Incidence; 4. Occupation Incidence.

1. *Sex Incidence.*—The majority of writers on plague agree that the sexes appear to be equally affected; and it will be interesting, therefore, to call attention here to the proportion of males affected and recorded in the Queensland statistics of plague.

The disease attacked nearly 3½ times as many males (359) as females (105). A similar incidence was observed in the Sydney epidemic of 1900, the male population being attacked more often than the female population in the proportion of about 4 to 1.

The nature of the occupations in which men engage, and especially those associated with the food trades (*see* "Occupation Incidence") necessarily expose men to relatively greater risk of contracting plague than members of the opposite sex, whose calling or duties do not take the latter into so ample fields for its origin. The fairly large proportion of attacks (64) among women engaged in household and domestic duties, including female servants and children at home, probably finds explanation in those domiciliary arrangements which make the kitchen, pantry, and larder peculiarly attractive to rat infestation.

2. *Age Incidence.*—On reference to Table V., Part I., showing the number of attacks and deaths distributed under various age groups, it will be observed that the heaviest incidence was on persons between the ages of 20 to 35, no less than 200 attacks with 80 deaths occurring at this period of young adult life. Sixteen attacks with 10 deaths occurred under 5 years of age, and the figures include 2 babies, 1 full-term, who died shortly after birth, the mothers also being fatal cases. The incidence on children up to the age of 15 years, 112 attacks (24 per cent.), with 55 deaths (40 per cent.), is noteworthy, as indicating the fact that though the prognosis in the cases of children is thought to be slightly better than in the case of adults, the diminished susceptibility to attack in

childhood is not so comparatively small as many observers have agreed. Indeed, Hankin ("The Journal of Tropical Medicine," 1st February, 1905, p. 40) draws special attention to the fact that some outbreaks of plague have chiefly attacked children; so much so, that the disease has been termed "Pestis Puerorum."

The number of cases, 64 (46 boys and 18 girls), which occurred among "school children," as shown in Table II., Appendix B, Part II., is so striking a factor that some explanation is needed to account for the incidence on this portion of the community. The sub-epidemic of plague which occurred at New Farm, a suburb of Brisbane, in January, 1907, was characterised by the number of children at school ages attacked. Of 16 cases which occurred within the "infected area," no less than 11 were school children (9 boys, 2 girls), of ages from 8 to 14 years. The first 7 cases reported were all schoolboys at home for the holidays, and playmates in constant association with each other up to the time of their respective illnesses. All were attacked at about one and the same time, and as mediate infection from one case to the other did not occur, there was apparently one source of infection common to them all. The whole 16 cases were directly associated with a general and fruit store, on which dead rats were found during the cleansing operations. The boys had been known to frequent a dilapidated shed containing produce and horse feed in the rear of the store, and a clear history of their handling dead rats in this shed was obtained. The above cases do not include another schoolboy, aged 14, living in the same district, but at some distance from the "infected area," and in no way associated with it or any of the children mentioned above, who died of bubonic plague two days after the onset of his illness at the beginning of the outbreak in New Farm, on 16th January, 1907. Infection in the latter case was traced to a produce store in the city, where four plague-infected rats had been found on 20th and 23rd December, 1906.

Reference will not be made again to the incidence of plague on school children, and it may, therefore, be mentioned here that the majority of Queensland children attending the schools go barefooted throughout the summer months. Cases did not often occur during the periods of school attendance, but at times of recess, when the roving habits of schoolboys on holiday would find full scope.

3. *Race Incidence.*—On the 31st December, 1901, the coloured alien population of the State was as follows:—

Race.	Population.	Percentage to Total Estimated Population.
Chinese	10,215	2·02
Pacific Islanders	9,951	1·97
Japanese	2,354	0·46
India and Ceylon	1,311	0·26
Other Asiatics	1,899	0·38
All Other Nationalities	480,214	94·91
Total Population	505,944	100·00

Exclusive of full-blooded Aborigines.

In consequence of the *Pacific Island Labourers Act* 1901, passed by the Commonwealth Government, some 3,295 Pacific Islanders or kanakas have been repatriated since October of 1906. The influence which is exerted on liability to plague by race has been set forth in the statistical data furnished in Table III., of Part I. The incidence on other races than whites has been but slight, the Chinese forming by far the largest proportion. The total number of plague cases (all races) for the whole State during the period of 1900 to 1907 was 464, with 195 deaths, a gross fatality of 42 per cent.

Included in the above are 22 cases among Chinese, a race amongst whom the fatality of plague is usually from 80 to 90 per cent., and, indeed, in our own Chinese cases 20 died out of the 22 attacked. If these and other aliens (including 2 aboriginals) be deducted, the corrected death rate for whites is 38·9 per cent.

4. *Occupation Incidence.*—The incidence on persons employed in the produce trade and the spread of infection by grain, fodder, &c., has already been noticed. The superior liability of dealers in grain and produce, store employees, grocers, butchers, fishmongers, cooks, hotel servants, stable-

men, &c., to contract plague may be attributed to the circumstance that the above and others associated with what has been called the "Food Trades," necessarily come into closer contact with rodent, and possibly insect life. The fact that food and fodder are liable to be contaminated by infection, and are likely to attract rats to the places in which such grain, produce, and foodstuffs generally are stored, receives emphatic corroboration from a study of the lists published as appendices showing the occupations of the persons attacked, and the incidence of plague-infected rats on the business premises. Of the 461 cases recorded in connection with occupation, it will be observed that no less than 166, or 36 per cent., were directly related with the food trades, or engaged in pursuits which exposed them to the risk of infection through the agency of rats.

As personal communication or mediate infection was not observed as a cause in any of the cases, the coming into contact in the places of business with a more or less large number of, and possibly infected, persons may be at once dismissed as an explanation of the prevalence of plague among this section of the trading community.

The instances to which attention may be more particularly directed, as affording the best indication of the amount of risk incurred by persons engaged in the produce and foodstuffs trades generally, are those in which a number of cases has been observed to occur in a single store or warehouse.

The Ipswich (1905), Rockhampton (1906), and New Farm, Brisbane (1907), sub-epidemics are instances in point.

The circumstances attending these outbreaks were briefly the following:—(1) All the seven patients were directly associated with the insanitary produce store at Ipswich in May, 1905. Dead and sick rats were found on the premises, a dilapidated shed, a week before the outbreak. (2) Eight cases, with four deaths, occurred at one warehouse (stocking foodstuffs, but no produce), situated in East street, Rockhampton, in April, 1906. Six of the sufferers were employed at the warehouse, and the seventh (fatal) and eighth cases were those of two schoolboys frequenting the store, and who rode in the cart removing rubbish from the premises during the cleansing operations. Dead and sick rats were found on and in the vicinity of the warehouse just previous to the outbreak. (3) Sixteen cases of bubonic plague occurred in January, 1907, at New Farm, Brisbane. All the cases were directly associated with the general and produce store where the first case was taken from.

In view of the fact that a certain class of the community is, from the very nature of its occupation, exposed to a greater risk of contracting plague than other sections of the trading public, the frequent occurrence of plague-infected rats on premises or places of business resorted to by the former class is highly significant. In this connection, it is instructive to compare Table II. (Appendix B) relating to the occupation of the patients with Table I. (Appendix A) showing the incidence of plague rats on the premises of the various trades. Of 954 rats found on bacteriological examination to be plague-infected, no less than 551, or 57·9 per cent., were found on premises connected with the food trades within the Metropolitan Area for the period 1900-1907. Of 313 plague-infected rats discovered during the first half of the year 1904, 88 were found during cleansing operations in one store in South Brisbane, and the produce stores were responsible for 111 out of the total of 313. The total number of rodents caught in produce stores during the 2½ months of 1904, when the special cleansing operations on grain and produce stores were carried out, was 6½ per cent. of the total number caught in the whole Metropolitan Area for the six months.

The risk of infection incurred by wharf labourers, rat-catchers, scavengers, disinfectors, packers of glass and crockery, and members of cleansing gangs is one of some practical importance, and here again, as in the class associated with the food trades or the produce business, the special liability to attack is proportionate to the contingency of contact with infection, either from plague-infected rats or in handling material likely to harbour rat-fleas. In several instances plague cases occurred in warehouses, the strictest search of which latter failed to reveal the presence of healthy, dead, or sick rats, the only important epidemiological factor being the packing material, straw, chaff, &c., which in at least five instances had been obtained from produce stores on which premises plague-rats had been found at a longer or shorter interval before the date of attacks respectively of the patients concerned.

In the eight cases of persons employed in rat-destruction operations and the cleansing and disinfection of plague-infected houses, two contracted infection through cutaneous lesions, two from rat-bite on the finger, and four while engaged in removing filth from beneath the incriminated store mentioned above in connection with the New Farm outbreak. That the risk to rat-catchers and scavengers is not great, and can be practically removed by the taking of ordinary precautions, is evidenced by the small proportion of attacks among the very large number of persons, often entirely uninitiated and careless of the routine precautionary measures adopted by the official cleansing staff, employed at various periods in cleansing and rat-catching operations within the municipality. The liability of doctors and nurses appears to have a direct relationship to the infectivity of each of the different clinical types of plague. While there is no clear instance of infection having been contracted directly from an ordinary bubonic case, the extreme infectivity of the primary pneumonic form in the few cases which have come under our observation is forcibly illustrated in the death of the two staff nurses of the Maryborough Hospital, and the elderly woman who attended the first case in the sub-epidemic at Maryborough in June, 1905.

The immunity of undertakers and persons concerned in the disposal and burial of plague corpses needs but a passing notice. No case occurred amongst this class. A wardsman of the Brisbane General Hospital died of plague in 1900, contracted at a *post-mortem* examination on a plague corpse. In all fatal cases the body was immediately surrounded by a sheet soaked in a solution of sublimate or carbolic acid, and placed in a coffin containing burned or slaked lime, the coffin in the grave also being surrounded by a layer of chlorinated lime at least 6 inches in thickness.

IV.—THE INFLUENCE OF INSANITARY CONDITIONS ON PLAGUE.

All observers have connected the occurrence and diffusion of plague with filth. By many plague is regarded as pre-eminently a filth disease, and more likely to occur in dirty than clean houses or districts. But while it can be shown that filth, *per se*, has but little influence, the measures intended to combat plague by effecting sanitary improvements, and by the raising of a higher standard of public and domestic hygiene, have been regarded, and very rightly regarded, as of the highest importance in the prophylaxis of the disease. These measures of sanitary reform are supported by many incidents in the history of the Queensland epidemics, and in the methods adopted for their suppression and restriction. Immediately following every special cleansing effort, undertaken voluntarily by the local authorities, or in compliance with official orders, a notable decrease in the number of attacks has been at once apparent. Quiescent periods of eight and nine months respectively followed the "Cleansing Orders" of 1902 and 1903. This cessation may have been merely coincidental in point of time with the removal of filth from, and the better scavenging of, the districts affected, but it was a "coincidence" which repeated itself again and again. Indeed, the most ordinary scavenging and removal of filth and garbage from the premises of infected places, and more especially from beneath the underspaces of floors of houses, were in the majority of cases sufficient to stay the spread of plague. In view of this fact, it cannot be argued that the ease by which this effect was produced was rather against than in favour of the proposition, that filth and insanitary conditions generally exercised a marked influence in the development and extension of plague. The cleansing operations which were undertaken not only in Brisbane, but also in every town that had been attacked by plague, were inevitably followed by a diminished incidence and a quickly following subsequent immunity.

That the *contagium morbi* is likely to be found in filth and accumulations of animal and vegetable refuse collected in dark, damp, and ill-ventilated places, either within or beneath dwellings and premises, and that such insanitary conditions are conducive to a stimulation of the vegetative life of plague organisms or the growth and development of the parasites which carry the latter, find illustration in the following examples:—

SERIES A.—ROCKHAMPTON EPIDEMIC, APRIL, 1906.

B.W., male, aged thirty-six years, carter. Attacked 7th April, 1906, with headache, vomiting, and pains in back. On the 8th instant he felt pain and noticed a swelling in the left groin. This was the third in a sequence of cases (8 attacks, 4 deaths) which occurred at the one warehouse, situated in East street, Rockhampton.

The above patient had been engaged in removing an accumulation of *débris* and rubbish from the basement floor in the back part of the warehouse premises, after the first case had been reported on the 3rd April. He helped to place the rubbish in a cart which he subsequently drove to the garbage depôt, some distance out of town, on the 5th April. A thorough inspection of the building by the local health officer, previous to the removal of the rubbish on the 5th instant, failed to reveal the presence of living rats or their dead bodies, though a clear history of rat infestation, and of carcasses of rats having been picked up in this part of the building a few days before the outbreak was obtained. The rear part of the warehouse premises was badly lighted, owing to the absence of windows, the warehouse employees often working by candlelight in the daytime. As already mentioned, a cartload of *débris*, dust, and rubbish was removed from this portion of the warehouse on the 5th April. That such rubbish did not contain the carcasses of rats, so far as could be observed, but some other infective material, or afforded a harbourage for rat-fleas, may be surmised from the fact that two boys, attending school, and aged thirteen and fourteen years respectively, accompanied the above patient, and rode in the cart on top of the rubbish *after it had left the warehouse premises*, on the 5th April, and while in course of the transit of the cart to the sanitary depôt, where the contents were disposed of by burning. Inquiry of the person who burnt this cartload of garbage at the depôt elicited the information that it did not contain the carcasses or skeletons of rats. One of the boys was attacked on the 7th April, the other on the 11th April. The latter died the same day, a *post-mortem* examination of the viscera showing the mesentery to be studded with numerous enlarged lymphatic glands. There was no superficial bubo. It was not ascertained whether this youth had partaken of any food while perched on the top of the contents of the cart. Both boys were accustomed to go barelegged and barefooted, and had numerous crusted sores on both legs and feet.

SERIES B.—BRISBANE (NEW FARM) EPIDEMIC, JANUARY, 1907.

1. (*a*) N.T., male, aged forty years; storehand. Date of attack, 22nd January, 1907. Died in Plague Hospital, 23rd January, 1907. Had been engaged on the 19th January (Saturday) and 21st (Monday) in removing a large accumulation of rubbish from beneath a general, produce, and fruit store, situated in James street, Kingsholme, New Farm, a suburb of Brisbane, previous to the outbreak there on the 16th January. This patient was the twelfth in a sequence of cases of plague which had occurred on, and in the immediate vicinity of, the infective store premises.

2. On the 21st and 22nd January, 1907, three (3) members of the cleansing and rat-destruction gang attached to the Department of Public Health, had been employed also in the removal of the rubbish above mentioned from beneath the store. The accumulation consisted of dust and *débris*, and putrefying animal and vegetable matter, &c., of a very offensive nature. During these cleansing operations it was noticed that fleas were very prevalent among this rubbish, and also among the bags containing the produce in the store, the men being repeatedly bitten. The store premises (*see* sketch, " Sub-epidemics") stood at the bottom of a hill, the ground sloping towards the store buildings, the soil beneath which latter was

highly polluted with the surface drainage of the surrounding houses. The underspaces of the floor were dark, damp, and dirty, conditions which instituted a dangerous nidus of infection. The particulars relating to the three latter cases are the following:—

(*a*) H.M., male, aged forty-nine years, member of cleansing gang, Department of Public Health. Attacked 24th January, 1907. Right femoral bubo. Severe case of the ordinary bubonic type. On the eighth day of his illness he coughed up some blood-stained sputum, which contained *B. pestis*. He recovered after a prolonged convalescence in the Plague Hospital.

(*b*) W.C., male, forty-two years, member of cleansing gang, Department of Public Health. Attacked 24th January, 1907. Right inguinal bubo.

This patient spat up some blood-stained sputum on the 7th day of illness, which yielded numerous *B pestis*. He became steadily worse, and died at the Plague Hospital on 31st January, 1907.

(*c*) H.J., male, fifty-two years, member of departmental cleansing gang. Attacked 26th January. Left femoral bubo. Made a satisfactory progress to recovery.

No further cases occurred among the other members of the official rat corps. It is a somewhat significant fact that the only members of the cleansing gang staff to contract the disease were the above three patients, who had been selected for this special work of cleansing, and who, in conjunction with the storehand, case 1 (*a*), were the only persons concerned in the removal of this particular rubbish from beneath the store premises.

Chief among the insanitary conditions that have been brought into association with plague in man or rats were those pertaining to municipal and local government administration. The amount of defective building construction admitting of rat-infestation was particularly observed in the case of the produce stores; and regulations having for their aim the remedying of these structural defects were made by me early in the year 1904. The hotels, restaurants, butchers' and grocers' shops, and other places connected with the food trades, where waste food material, and easy access to the same, served as a special attraction for rats, were also the subject of special by-laws relating to frequent and proper scavenging. The installation of small movable galvanised-iron bins or boxes, fitted with covers, in every house and workplace throughout the municipalities of North and South Brisbane, and the strict enforcement of the by-laws compelling householders and others to place waste food and other kitchen refuse in such receptacles, did much to reduce the incidence of rat-plague on those areas.

In Queensland, the dwellings are, for the most part, constructed of wood, roofed with galvanised-iron as a protection against hail-storms, and built on piles several feet from the ground as a protection against the ravages of white ants. The space beneath the house, often an ample one, was in many instances barred off and roughly shut in as a fowl-yard, a stable, a lumber-room for the storage of grain or fodder, and, in not a few cases, a living room for the inmates of the house. In this way rats were brought into the closest association with the members of the household.

Apart from any theoretical consideration of the influence of insanitary conditions generally on the spread of plague, the dangers of defective drainage and sewerage were early recognised as a matter of practical observation. The defective condition of, and numbers of untrapped connections opening into, the sewers laid down by the municipal authorities in the early days of Brisbane have already incidentally been mentioned. A good idea of the type of drainage works alluded to is shown in the rough pen and ink sketch attached. (Appendix D, Part II.)

The premises upon which this "specimen" was unearthed had constantly been infested with rats which, at the first note of warning, as suddenly made their exit from as they gained entrance to the building and the surrounding places. To the free-and-easy methods of sanitation adopted by former " civic fathers" many of the difficulties of rat-destruction experienced by the officers of the Health Department in latter years were due. The influence of insanitary conditions, then, may be summed up by stating that the infectiveness of districts and localities was chiefly if not entirely engendered by the infectiveness of the particular premises which stood within them; or, in other words, the incidence of plague on certain localities was mainly due to the opportunities for infection afforded by the presence of plague-infected rats on premises structurally defective within, or environed about by insanitary conditions acting as an attraction to, or affording facilities for, rat-infestation.

V.—INFLUENCE OF SEASON.

The most striking factor in the history of plague in Brisbane is the cyclical character of its annual increase and decrease. The development and decline of the eight epidemics of plague in Brisbane during the successive years 1900-1907, inclusive, are shown in the Stratigraphic Chart in the Appendix to Part IV. Written after the events have taken place, it is now possible to read the charts as one continuous record. Upon the charts have been entered week by week the advent of plague in April, 1900, to the end of June, 1907; the number of cases in man and rats; and various meteorological factors including the grand mean temperature, the grand mean vapour tension, the rainfall, the relative humidity, and the frequency of the west winds.

A consideration of the chart as a continuous record will show the epidemic curve to be in fairly close agreement with that of the epizoötic curve. Both reach their maxima in the autumn months of April or May, are lowest in the winter months of June or July, and are normal or absent altogether during the spring and early summer months of the latter half of the year. Indeed, with the exception of a sporadic case or two both in man and rats, an almost straight line, indicating the period of quiescence, may be ruled from the month of July to the end of December in many of the years under review. This quiescent period often extended to the end of January of the following year. It will be observed that the epidemics are synchronised closely with the epizoötics for each successive year, the cases in man being invariably preceded and accompanied by the epizoötic amongst rats, the sporadic cases in the latter beginning a month sooner and lasting a month or two longer than the former.

The cases in human beings are observed to begin during the hot and moist or "muggy" season of the year—the end of January and the month of February—reach their limit about May, and notably decline at the beginning of June when the colder weather with the dry westerly winds set in.

No epidemic in Brisbane, with the exception of that in 1900 when cases continued to appear in sporadic form up to December, lasted longer than about twelve to fourteen weeks.

The coastal climate of Queensland, with its even temperature and comparatively high humidity, is probably more constant than any other place within the same isothermal lines.

The *grand mean temperature* for the month of February is about 77 degrees in each of the recurrent epidemic years, falling to about 65 degrees in June and July, and then steadily rising to the maximum of 77 degrees towards the end of the year in December.

The following table shows the grand mean temperatures for certain months in the years 1900-1906:—

Table Showing Grand Mean Temperature.

Years.	January.	February.	May.	June.	December.
1900	74·6	77·3	63·9	60·9	77·1
1901	77·4	75·3	64·2	57·6	76·7
1902	79·7	78·4	64·7	63·0	79·4
1903	80·0	79·1	64·2	59·5	77·2
1904	76·8	78·0	64·4	57·4	76·3
1905	77·3	77·1	65·3	59·0	76·9
1906	77·3	76·1	64·9	61·6	75·4
Average for 7 years F.	77·6	77·3	64·5	59·9	77·0

The curve of the *grand mean vapour tension* will be observed to follow a remarkable parallel course to the grand mean temperature curve, always sinking or rising with the latter. From about 0·633 in January, and 0·641 in February, of each year, the curve descends to 0·432 in May, and 0·350 in June; to rise again with the temperature curve during the last half of the year. The figures are shown in the following statement:—

TABLE SHOWING THE GRAND MEAN VAPOUR TENSION.

Years.	January.	February.	May.	June.	December.
1900	0·648	0·676	0·414	0·364	0·608
1901	0·625	0·630	0·452	0·327	0·568
1902	0·604	0·628	0·412	0·377	0·659
1903	0·590	0·645	0·423	0·323	0·604
1904	0·618	0·562	0·471	0·322	0·647
1905	0·700	0·682	0·422	0·347	0·622
1906	0·647	0·666	0·430	0·391	0·558
Average for 7 years ...	0·633	0·641	0·432	0·350	0·609

The *relative humidity* (grand mean) is about 65 per cent. (sat. = 100) in January, 66 per cent. in February, 65 per cent. in June, 54 per cent. in September, and 61 per cent. in December.

The figures for six successive years are given below:—

TABLE SHOWING THE RELATIVE HUMIDITY.

Years.	January.	February.	May.	June.	December.
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
1900	76	72	70	68	66
1901	66	71	75	69	64
1902	62	66	68	69	68
1903	60	67	72	65	56
1904	58	50	73	60	62
1905	67	66	62	60	58
1906	62	68	62	64	55
Average for 7 years ...	65	66	69	65	61

It will be noticed, on reference to the chart, that a fall in the mean temperature and vapour tension at the beginning of the year was generally followed by a rise in the number of cases of plague in man, and an increase in the number of infected rats.

West Winds.—During the winter months of the year—June, July, and August—the westerly winds blow in Brisbane for several days at a time. This wind has a remarkable drying effect upon all organic life. It is Nature's scavenger; retarding the growth of insect life, and exerting a powerful desiccating influence on all dead organic waste matter left exposed to its action. A notable fall in the epidemic and epizoötic curves was constantly observed to follow shortly after the prevalence of these winds. The number of days in each month of the years 1900-1906, inclusive, on which the west winds were observed to blow are given in the following table:—

TABLE SHOWING PREVALENCE OF WEST WINDS.

Years.	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.
1900	0	1	3	11	13	16	21	16	6	2	1	0
1901	0	0	0	7	3	16	19	7	3	5	0	0
1902	1	0	1	0	3	5	8	7	10	5	1	4
1903	1	1	0	5	15	18	21	3	9	2	1	5
1904	1	3	0	3	5	8	8	5	5	0	0	0
1905	0	0	1	3	15	14	14	10	19	13	0	3
1906	0	0	3	4	6	10	18	13	6	4	4	2
Average No. of days for 7 years ...	0·43	0·70	1·14	4·71	8·57	12·43	15·56	8·71	8·29	4·43	1·00	2·00

Rainfall.—Moderate rainfall during the progress of an epidemic was frequently followed by an increase of cases in human beings. Heavy and continuous rains appeared to be unfavourable to the epidemicity of plague, but a moderate fall with heat when plague was current so constantly predicted the arrival of a fresh case at the Isolation Hospital as to pass into a common saying, "More rain, more plague," among the nurses at the institution.

The rainfall in inches and the number of wet days for each month during the period 1st January, 1900, to 30th June, 1907, are recorded in the table below:—

TABLE SHOWING RAINFALL AND NUMBER OF WET DAYS PER MONTH FOR EPIDEMIC YEARS 1900-1907.

Months.	1900.		1901.		1902.		1903.	
	Rainfall.	Wet Days.	Rainfall.	Wet Days.	Rainfall.	Wet Days.	Rainfall.	Wet Days.
January	6·511	12	3 429	6	1·378	10	1·310	9
February ...	5·184	11	2·962	13	2·671	7	5·348	14
March	3·365	16	11·696	18	0·630	2	4·792	17
April	1·378	7	3·104	10	0·173	7	1·331	9
May	5·448	15	2·275	11	0·474	3	11·815	15
June	2·678	11	3·293	7	0·057	2	0·730	5
July	4·363	8	1·310	6	0·553	9	5·556	14
August	0·794	3	3·710	9	0·977	10	3·835	16
September ...	1·521	7	1·296	7	1·296	6	4·733	8
October	0·141	4	3·252	9	3 421	8	3·653	13
November ...	2·478	7	1·405	10	2·589	11	3·982	12
December ...	0·548	9	0·748	4	1·818	11	2·187	7

Months.	1904.		1905.		1906.		1907.	
	Rainfall.	Wet Days.	Rainfall.	Wet Days.	Rainfall.	Wet Days.	Rainfall.	Wet Days.
January	2·647	12	9·094	12	4·161	10	2·691	17
February ...	0·768	5	2·631	10	12·708	15	5·233	14
March	7 070	19	2·651	10	4·849	23	5·316	9
April	7·234	19	4·503	14	0·449	2	0·445	11
May	4·039	19	1·100	12	3·233	6		
June	0·589	5	0·388	9	1·375	11		
July	1·484	7	0·275	3	0 220	5		
August	0·525	8	0·652	6	4·211	6		
September ...	1·590	5	1 321	4	3 477	14		
October	1·281	10	2·219	8	3·806	13		
November ...	2·355	7	3·630	9	1·068	8		
December ...	3 647	8	8·295	11	3 279	12		

That plague is particularly liable to recur in periodical outbreaks is a fact which has been established in many places where the disease is known to be endemic. It is also equally true that plague has a tendency to become epidemic only at certain seasons of the year. "This season," says Simpson in his "Treatise on Plague," "may vary somewhat in different localities, but it is nearly always the same in the same locality, and has a tendency to become earlier the further south it occurs."

This seasonal periodicity has been observed in the Australian epidemics of plague. In Brisbane the epidemic reached its maximum in the month of May. In Sydney, which is situated much further south, the months of March and April furnished the largest number of cases during an epidemic year. In the more northern towns of Queensland—*i.e.*, Maryborough, Rockhampton, Townsville, and Cairns—the months of epidemic prevalence have been June, July, August, and September respectively.

Towards the latter end of January and during the whole of the month of February and part of March, the weather in Brisbane is hot, moist, and "muggy." The temperature is high and equable and the atmosphere saturated with moisture—hot-house conditions favouring a prolific vegetable growth. The above period, especially the month of February, is the rainy season of the year, the highest annual and monthly mean falling on the month of February for a period of forty-six years past. It is a time of the year when Nature is most prodigal of her flora and fauna, a luxuriant and manifold vegetation and a notable increase in insect life abounding.

On the other hand, during the winter months, the period of freedom from plague, vegetation is at a standstill and insects hibernate. Whether the plague bacillus finds in seasonal variation a change of environment suitable or unsuitable to its survival or not, there are certainly grounds for inferring that any influence exerted by meteorological conditions upon the higher forms of vegetable life extend to the lower forms, even to those lowly minute algoid plants known as "bacteria."

Before dealing with the epidemiological observations which may come into consideration in connection with this question of seasonal influence, it may be as well to advert here to certain axioms, which, I think, are capable of explanation by the theory of seasonal or climatic variations. In this connection attention may be directed to the following brief summary of facts which have been more or less definitely established:—

(1) Seasonal variation of plague is uniformly constant in infected places where the disease is endemic.

(2) The season of the year has a very powerful influence on the prevalence of epidemic plague and the duration of the epidemic.

(3) Great epidemics of plague are not universal, but prevail over limited areas, the localisation depending on the distribution of the animal and insect life concerned in the spread of plague.

(4) That geographical distribution and season have a direct or indirect influence on the number, variety, and prevalence of animals or insects capable of acting as agents or media in connection with plague and other infectious diseases.

(5) That season, with its meteorological variation, has an influence on the survival, growth, and virulence of the bacterial flora, including the plague organism.

(6) That there is an adaptation, on the one hand, of the plague bacillus to grow within the human or animal organism, and an adaptation, on the other hand, of the bacillus to survival in a more or less virulent condition in the external and particular environment of an infected locality; this external environment varying from place to place and from season to season.

Many theories have been put forward by writers as to the cause of the seasonal variation of plague, but, up to the present, no causal association has been established which could harmonise the conditions particularised above with the prevalence of the disease. The question still remains one of the many unsolved problems of plague. The striking recurrence of the rise and fall of the case attacks in successive years, cannot be the mere coincidence or product of chance which certain writers would seem to agree upon.

Beyond a mere passing inference to the observed relationship between season and plague, the majority of writers are content to dismiss the subject with a statement to the effect that climate and season exercise a small, but not very important, influence on the origin and spread of a plague epidemic. Others, again, have suggested that the seasonal periodicity of plague may be due to varying hygrometric conditions and temperature of the soil; the influence of weather on the habits and movements of the population—*e.g.*, cold nights and heavy dews confining people to their houses, or excessive rainfall interfering with remedial measures; the "acclimatisation" theory; the influence of seasonal meteorological variation on the vitality of the plague bacillus outside the animal organism; and the seasonal increase of plague among rats. In an attempt to analyse some of the phenomena observed in connection with season, certain meteorological statistics have been graphically set forth in the chart prepared by me covering the eight successive and epidemic years, 1900-1907 inclusive, of plague in Brisbane. (*See* Chart, "Seasonal Prevalence of Plague.")

In its way the chart affords an excellent example of the cyclical and seasonal characteristics of most great epidemics of plague. The most casual glance will show the rise and fall in each successive year. There is, first of all, a gradual beginning in the month of January or February, then there is a more rapid rise, which reaches its maximum in May; a sudden fall in June, and finally a period of freedom, punctuated in some years by one or two sporadic cases in man and rats.

At the present time we possess a fairly complete laboratory knowledge of the bacteriology of plague, but comparatively little is known of the behaviour of the *B. pestis* in external Nature. Art may imitate Nature, but can never replace it. As Rosenau (Bulletin No. 4, Hygienic Laboratory, Washington) very pertinently remarks: "The bacillus of plague does not exist in Nature on sterile glass cover slips, nor yet in the desiccator over concentrated sulphuric acid. . . We attempted to imitate Nature; but we cannot imitate all the conditions under which the organism may exist in Nature, and we ought not, therefore, to apply the experience of the laboratory too literally to the life history of the plague bacillus outside of the body. We may determine with fair certainty the length of time the bacillus may live under given conditions, but these conditions are more or less arbitrary, and to a certain extent artificial." It would thus appear that two different sets of attributes are necessarily involved in the capacity on the part of specific bacteria for growth within the tissues of living animals, and for survival in external conditions outside the human or animal organism. The question is one of great epidemiological importance, and further investigation should be conducted more along the lines of practical exploitation in the open rather than in the somewhat narrowed region pertaining to laboratory investigation.

Under the latter, the bacteriologist rarely realises this limitation, from his point of view; or that the many factors that influence the life of specific bacteria in the outer world—*e.g.*, the strains or races which have different powers of resistance to external conditions, the neutralising effect of contaminating organisms, the tendency to adaptability, the variation in virulence, and the chances of escape from the infected organism—are always, as a matter of fact, epidemiologically operative. Further reference will be made to this observation in dealing with the subject of destruction of rats by the application of bacteriological methods, and notably by the Danysz bacillus.

The influences of light, temperature, moisture, and drying are well understood from the many laboratory experiments carried out by bacteriologists to determine the viability of the bacillus of plague. The influence of temperature would appear to be in direct proportion to the amount of moisture or humidity of the atmosphere, the plague bacillus requiring temperature and moisture for its active existence. The *Bacillus pestis*, further, usually dies quickly when dry, but is not as sensitive to temperature when kept moist. Here, then, in Brisbane, during the early part of the year the plague organism finds, in the exceptional circumstances of a high and equable temperature and humidity of the atmosphere, climatic conditions highly favourable to its development and survival. On the other hand, the incidence of plague, both in man and rats, diminishes or altogether ceases during the winter months. There is no reason to believe that the plague bacillus cannot and does not exist in soil, especially in moist earth in dark and warm places. It is true that it does not undergo endogenous spore formation, and cannot be kept alive at any temperature in dry earth, but the conclusion that the *Bacillus pestis* is a frail saprophyte is no doubt founded on the assumption that no definite cultural method can be relied upon to reveal the presence of plague bacilli when mixed with a large number of contaminating organisms.

Defective technique, rather than direct demonstration by bacteriological investigation, would appear to practically sum up our present knowledge with regard to plague in the soil.

It has been suggested by some observers that the seasonal variation of plague may be due to seasonal increase of plague among rats.

It may be mentioned here that our experience leads us to believe that rats in Queensland breed all the year round, a slight increase, perhaps, being noticeable in the months of June, and towards the end of December and beginning of the year. A more feasible explanation of the seasonable variation of plague may perhaps be found in the prevalence and activity of insect life, notably fleas, and the development of the egg, larva, and pupa of fleas under certain environmental conditions. As an instance of certain seasonal conditions upon the prevalence and activity of insect life, it may be interesting to note that, in the months of January and February, fleas were very prevalent in certain of the epidemic years, and markedly so in that of 1907. These fleas appeared in swarms about the gardens, sandy patches, outhouses, and even within the dwellings. Numerous complaints were made of the nuisance, and remedial measures were devised to mitigate it. A similar nuisance was also observed by the health authorities about this time at Sydney, New South Wales, and Perth, West Australia.

A second interesting fact as bearing on the question of the seasonal prevalence of fleas may be mentioned in the circumstance that during an epidemic of plague little difficulty was experienced in collecting the fleas from off the rats, but that towards the end of the epizoötic the number of fleas on rats was small, the rats appearing to be exceptionally free of fleas as soon as the quiescent season was reached. Our experience in this connection was confirmed by a similar observation made by Dr. Frank Tidswell, of the Department of Public Health, New South Wales [*cf.* Tidswell, F., Report of the Board of Health on a Second Outbreak of Plague at Sydney, 1902, p. 74, par. 291]. Fleas are intolerant of wet, and are driven into the houses during rainy weather. This may explain the increased incidence of plague on human beings after moderate rainfall during the epidemic.

VI.—LOCALISED OR SUB-EPIDEMICS.

That infectivity attaches to localities, and, in some instances, to premises, is illustrated in the following brief descriptions of sub-epidemics.

I. IPSWICH OUTBREAK, 4TH TO 7TH MAY, 1905.

All *seven* patients were directly associated with a produce store, situated in one of the main streets of the town. The proprietor, his wife, and two assistants were engaged in mixing chaff. Dead and sick rats were found in the store—a dilapidated shed—a week before the outbreak. The floor of the shed was inches deep in *débris*, and fleas were very numerous in this litter. A man, who visited the store for the purpose of purchasing chaff two days before the first case occurred, took sick on the same day as the latter. He sampled the chaff with his hands, but refused to buy it on the ground that it was " stale looking."

All the above persons, including another, a carpenter, working in the store, drank from a tap in the shed, using their unwashed hands as a drinking utensil. Four of these were taken ill with abdominal pains, vomiting, and headache. The seventh case, a boy, employed in hawking fruit obtained at the store, died on 9th May at his own home.

The outbreak was limited to these seven cases.

II. MARYBOROUGH CASES—MAY-JUNE, 1905.

An outbreak of plague, attended with the deaths of eight persons, *including five of one family*, occurred at Maryborough, a city of 12,000 inhabitants, distant 167 miles by rail from Brisbane. A full account of the outbreak will be found in Appendix D, Part II.

Briefly, the facts are as follows:—

Five members of a family of seven children, living in a state of poverty and neglect, were seized within a few days of each other with acute gastro-intestinal symptoms. The first case was that of a boy of seventeen years, employed as an assistant in a fruiterer's shop in the city where plague-infected rats had previously been obtained. Four of the children, whose ages ranged from seven to fifteen years, were taken ill on the 28th May, with headache, pyrexia, severe vomiting, and pains in the cardiac and epigastric regions. They were removed to the Maryborough District General Hospital, where they died in a few days with more or less marked pulmonary symptoms.

In all of the above cases the variety of plague partook of the nature of a general infection, and in no case was there any palpable evidence of enlarged lymphatic glands.

With the exception of the first case, a boy of seventeen, who died at his own home on the 25th May, pulmonary symptoms did not develop until after admission of the remaining patients to the General Hospital.

A careful and prolonged search for rats in the home of the family was attended with negative results. A subsequent search for rats by an experienced staff of rat-catchers in the vicinity of the infected house particularly, and about the town in general, revealed no evidence of plague-infection among the rats, though large numbers were obtained, many of them being of exceptional size. All of the rats bacteriologically examined proved to be of the *Mus decumanus* species.

Two staff nurses of the Maryborough General Hospital, who attended the cases admitted to the latter institution, died of *primary plague pneumonia*. A third nurse, who sickened, recovered.

The association and distribution of the cases as well as the time incidence of the attacks are given below:—

GROUP A.

No. of Case.	Name.	Age.	Date of Attack.	Date of Death.	Remarks.
1	John O'C. ...	17 years ...	19 May, 1905	25 May, 1905	Died at his own home.
2	Mrs. E. ...	49 ,, ...	28 ,, ,,	31 ,, ,,	Died at her own home; a neighbour who attended Case 1.
3	Ellen O'C. ...	7 ,, ...	28 ,, ,,	31 ,, ,,	Died at Maryborough General Hospital.
4	James O'C. ...	15 ,, ...	28 ,, ,,	31 ,, ,,	,, ,, ,,
5	Ritchie O'C. ...	10 ,, ...	28 ,, ,,	2 June ,,	,, ,, ,,
6	Mary O'C. ...	3½ ,, ...	28 ,, ,,	3 ,, ,,	,, ,, ,,
			GROUP B.		
7	Nurse B. ...	22 years ...	3 June, 1905	6 June, 1905	Died at Maryborough General Hospital.
8	Nurse W. ...	28 ,, ...	5 ,, ,,	12 ,, ,,	,, ,, ,,
9	Nurse E. S. ...	28 ,, ...	10 ,, ,,	...	Discharged, convalescent, 23rd June.

III. ROCKHAMPTON OUTBREAK, APRIL, 1906.

Eight cases of bubonic plague with four deaths occurred at one warehouse situated in East street, Rockhampton. The first case was notified on the 3rd April.

Six of the patients were employed at the warehouse, and the seventh and eighth patients were those of two schoolboys, who rode on a cartload of rubbish removed from the warehouse prior to the cleansing operations. One of these boys died within a few hours of attack; the other recovered. The man who drove the cart also developed plague. All eight cases occurred within a period of eight days. At the time the cleansing operations were undertaken by the Health Department—after the first case on the 5th April—a strict search of the warehouse premises failed to reveal the presence of live or dead rats. No produce was stored in the infected premises.

It was subsequently ascertained that dead and sick rats had been seen on the warehouse premises prior to the date of the first case. There was also evidence to show that the first two patients had handled dead rats. A number of dead rats in a state of decomposition too far advanced to admit of a definite diagnosis being made as to the cause of death, were also discovered in a store adjacent to the warehouse and in an adjoining lane. It is highly probable that these rats died of plague.

The particulars of the cases are as follows:—

No. of Case.	Initials.	Sex.	Age.	Onset.	Result.	Remarks.
			Years.			
1	E.W.	Male ...	18	3 April ...	Died 7th April	A clerk employed in the warehouse.
2	T.M.	,, ...	15	5 ,, ...	Recovered ...	A messenger ,, ,,
3	W.B.	,, ...	36	7 ,, ...	,, ...	A carter ,, ,,
4	S.T. ...	Female	39	7 ,, ...	,, ...	A charwoman ,, ,,
5	G.W.	Male ...	27	10 ,, ...	Died 19th April	A storeman ,, ,,
6	P.J.E.	,, ...	26	11 ,, ...	Died 15th April	A storeman ,, ,,
7	H.R.	,, ...	13	11 ,, ...	Died 11th April	A schoolboy who rode on top of cartload of rubbish removed from the warehouse on 5th April. Cart driven by Case 3.
8	T.H.P.	,, ...	14	12 ,, ...	Recovered ...	Rode with Cases 7 and 3 on cart.

IV. NEW FARM OUTBREAK (BRISBANE), JANUARY, 1907.

Sixteen cases of plague of the bubonic type occurred in January, 1907, at Kingsholme, New Farm, a part of the city of Brisbane within the municipal boundary. The outbreak was a typical example of *local place infection*, the "infected area" being a small block bounded by four streets.

All the cases were directly associated with the general and produce store (Garnett's) situated in James street, New Farm.

GROUP I.

Case No. 1.—G. A., aged 14 years, a schoolboy, residing with his father, a general storekeeper and produce merchant, James street, Kingsholme, New Farm. Ill two days previous to admission to the Brisbane Hospital for Sick Children, on 16th January, with cerebral symptoms. A brother of this boy died in the Children's Hospital on 13th January with symptoms of cerebro-spinal meningitis. The produce sold at the store was obtained from a produce store in the city, where four plague-infected rats were obtained between the 20th and 23rd December, 1906.

Case No. 2.—McG. J., aged 13 years, schoolboy, residing with his parents in close vicinity to Case No. 1. Admitted to Children's Hospital on 16th January, with cerebral symptoms, vomiting, and abdominal pain.

Case No. 3.—N. J., aged 12 years, schoolboy, residing with his parents at James street, immediately opposite to the store (Case No. 1). Admitted to Children's Hospital on 17th January, with cerebral symptoms and vomiting.

Case No. 4.—N. L., aged 17 years, brother of N. J., Case No. 3, and residing same place. Admitted to Children's Hospital on 17th January with fever, femoral adenitis, and delirium.

Case No. 5.—E. J., aged 10 years, schoolboy, residing in vicinity of Cases Nos. 3 and 4. Admitted to Children's Hospital 17th January with headache and abdominal pain.

Case No. 6.—N. W., aged 13 years, schoolboy, residing with parents, Brown street, New Farm, in vicinity of above-mentioned cases. Admitted to Children's Hospital 16th January with headache, pyrexia, and abominal pain.

Note.—Cases Nos. 1 to 6 were all schoolboys, at home for the holidays. The boys were playmates, and were in constant association up to the time of their illnesses. There was, apparently, one source of infection common to all.

It was not until after admission to the Children's Hospital that the real nature of the disease revealed itself. Patients 1 to 4 developed glandular swellings and symptoms suggestive of plague. In these cases the bacteriological investigation proved beyond doubt the presence of *B. pestis*. Cases 5 and 6 developed no glandular swellings and were kept under observation as "suspect" cases.

All the boys had been in and out of the general store, handling goods, and playing in the shed where the produce was kept. In the latter, twelve skeletons of rats were obtained during the cleansing operations. An active search for rats in the general store proved futile, and only one rat (alive and healthy) was caught.

GROUP II.

Case No. 7.—M. W. F., aged 14, schoolboy, residing with his parents at Browne street, at rear of the yard of general store. Attacked 18th January. Removed to Plague Hospital on 20th January. The boy noticed a cat bringing rats and mice from the store premises a week before he was taken ill. This cat, he states, had numerous fleas upon it. This patient died in hospital on 25th January.

Case No. 8.—M. M., female, aged 75 years, grandmother of Case No. 7, and resided with him. She was attacked on the 19th January, and died at her own home on 21st January. Type: bubonic. Had nursed Case No. 7 before his removal to hospital. She obtained goods at the store (Garnett's).

Case No. 9.—F. R. A., schoolboy, aged 8 years, living with parents Hawthorne street, New Farm, at rear of store. Case reported after death. Attacked 19th January. *Post-mortem* examination revealed presence of *B. pestis*. Dead rats found in yard of the premises.

Case No. 10.—G. R., schoolboy, aged 8 years, residing Browne street. Cousin of patients Nos. 1 and 11. Was in constant association with Cases Nos. 1 and 6. Attacked 19th January.

Case No. 11.—G. E., schoolgirl, aged 9 years, sister of Case No. 1. Attacked 22nd January. Removed to Plague Hospital 23rd January.

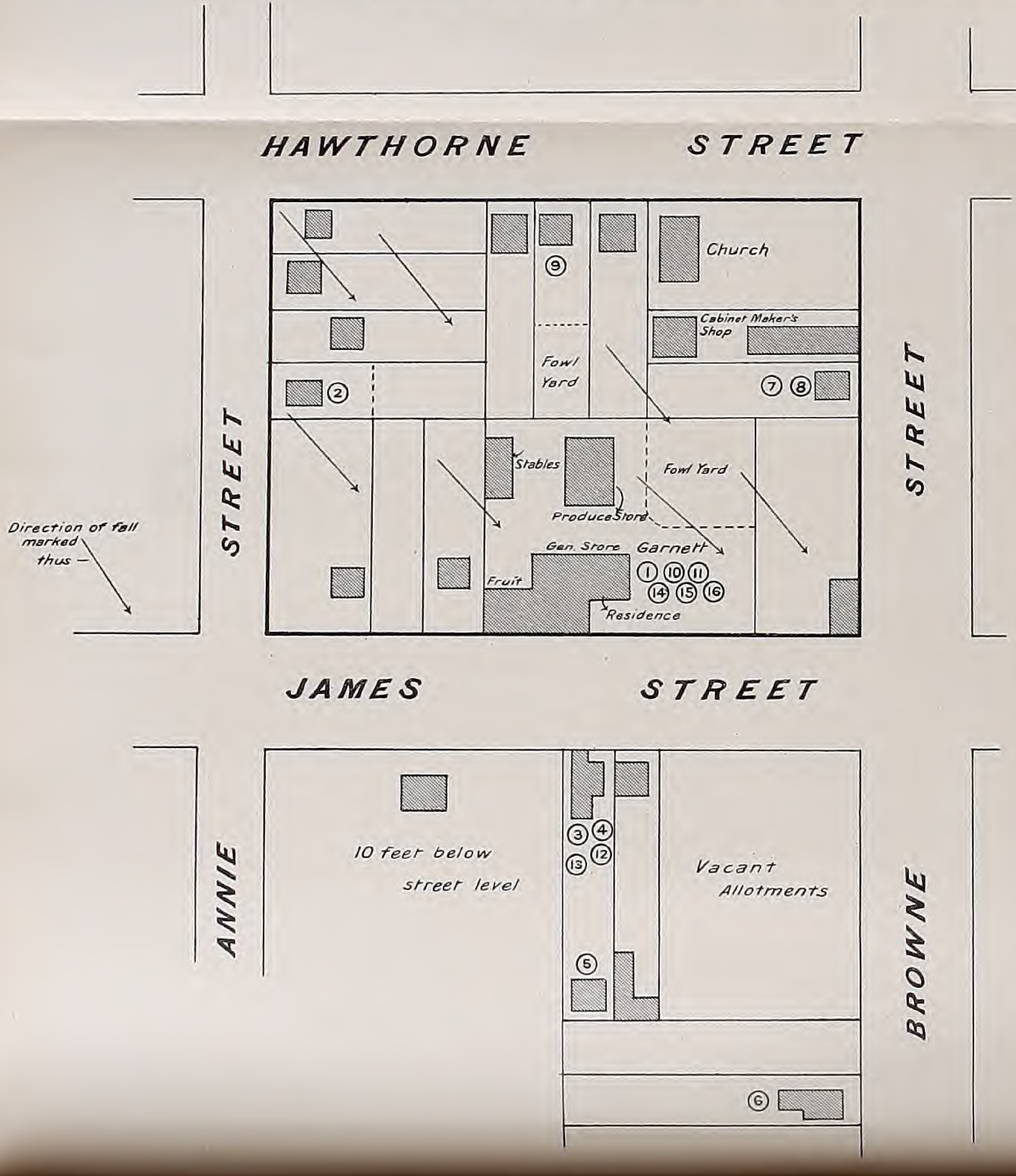
Case No. 12.—N. J., male, aged 40 years, James street, New Farm, opposite Garnett's Store. Father of patients Nos. 3, 4, and 13. Had been recently employed at the store. Attacked 22nd January. He found and burned a dead rat discovered in the store a week before onset of illness. He had also been employed in cleansing operations at the store at the beginning of the outbreak. Handled the produce and other goods. Removed Plague Hospital 23rd January, and died there on the 30th January.

Case No. 13.—N. L., female, aged 14 years, residing with her parents James street, New Farm. Sister of Cases Nos. 3 and 4, and daughter of Case No. 12. Attacked 20th January.

GROUP III.

Case No. 14.—H. M., male, aged 49 years, a member of the Cleansing Gang, Health Department, working in the "infected area." Attacked 24th January. Removed Plague Hospital 26th January.

Plan of Infected Area, Brisbane, January, 1907.



DISTRIBUTION & SEQUENCE OF CASES

Nº	Initials	Sex	Age	Occupation	Date of Attack
1	G. A.	M	14	Schoolboy	January 14
2	Mc C.	M	13	,,	,, 16
3	N. J.	M	12	,,	,, 16
4	N. L.	M	10	,,	,, 16
5	E. J.	M	10	,,	,, 16
6	N. W.	M	13	,,	,, 15
7	M. W. F.	M	14	,,	,, 14
8	M. M.	F	75	Household duties	,, 19
9	F. R. A.	M	8	Schoolboy	,, 18
10	G. R.	M	8	,,	,, 20
11	G. E.	F	9	Schoolgirl	,, 22
12	N. J.	M	40	Store hand	,, 22
13	N. L.	F	14	Schoolgirl	,, 25
14	H. M.	M	49	Cleansing gang (Dept of P. H.)	,, 24
15	W. C.	M	42	,,	,, 24
16	H. J.	M	52	,,	,, 26

Case No. 15.—W. C., male, aged 42 years, member of the Health Department Cleansing Gang, employed in the "infected area." Attacked 24th January. Removed Plague Hospital 26th January. Died 31st January.

Case No. 16.—H. J., male, aged 52 years, also member of the Departmental Cleansing Gang employed in cleansing operations in the "infected area." Attacked 26th January. Removed Plague Hospital 28th January.

Note.—The above patients (14), (15), and (16) were men employed by the Department to cleanse the infected area.

On the 21st January the above three men were employed in removing a large accumulation of filth—putrefying animal and vegetable matter—beneath the general and fruit stores. The men, therefore, took ill before the cleansing operations had time to become effective. With other members of the gang they were employed on the 22nd instant in removing the produce from the shed, and two of them (Cases 14 and 15) complained to the foreman of the gang of feeling sick. They were absent from work on the 23rd and 24th January, and were reported as plague cases on the 26th January.

Case 16 was not attacked until the 26th January; he was removed to hospital on the 28th January.

At the time the above, in association with other members of the cleansing gang, were employed in handling the produce in the out-shed, it was noticed that fleas were very numerous in this shed and also in the yard belonging to the premises.

It is a curious coincidence that of the seven men and the ganger employed in cleansing the infected area at New Farm only three of the men—Cases 14, 15, and 16—were occupied in removing the filth from beneath the store and fruit shop, and these were the only members of the gang to contract plague.

The invasion stages also point to the 21st January as the date of onset in at least Cases 14 and 15. Case 12, a man of forty years, living opposite the store, was also employed previous to his attack on the 22nd January in removing part of the material from beneath the store.

All of the above four patients developed secondary pulmonary phenomena while in the Plague Hospital, Case 12 dying on the 30th January, and Case 15 on the 31st January.

The accompanying plan shows the distribution and sequence of the cases within the infected area.

VII.—PLACE INFECTION.

The following notes on the etiology of the nineteen cases comprising the small outbreak of 1903 (metropolitan area) establish the fact that the infectivity of the fifteen premises (dwelling or workplace) furnishing the nineteen cases was entirely due to the presence on them of plague-infected rats. Every case (with the exception of two living on premises which showed signs of rat infestation though no live rats or carcases were actually obtained) was closely associated with plague-rats.

Case No. 1.—Clerk by employment, but out of work for some time. Lives in healthy spot on Rutledge's Hill, Paddington. Has been in the habit of visiting Case No. 3, at the shop where the latter works in Edward street.

Case No. 2.—Works as an engineer on premises near the river bank at Petrie's Bight. A sewer opens into the river near the works, and it was along the line of this sewer that a chain of cases took place in 1901 and 1902. A plague-infected rat was obtained from the premises during the cleansing operations.

Case No. 3.—Grocer's assistant. Works at a shop in Edward street. Sickened a day or two before Case No. 1, who was probably infected from the same source.

Case No. 4.—Lives in a house in the yard of a large flourmill. Large sheds, containing stored maize and wheat, adjoin the house. Rats are always plentiful, owing to abundance of feed. One large shed, containing maize, has its floor only 3 or 4 inches from the soil, and when this was taken up the carcases of several hundred rats were unearthed. A week before the child sickened the flooring of the kitchen had to be taken up in places to remove the dead bodies of decomposing rats. The father says he has often seen rats in a stupid and dazed condition near the granary, and he has killed them with a stick on several occasions. This case appears to have developed the disease in the submaxillary glands first, as inguinal and femoral buboes did not make their appearance till the illness had lasted five or six days. Plague was not suspected by the medical attendant till the involvement of the inguinal glands became apparent.

Case No. 5.—Worked in the basement of a Queen-street premises. There were numerous traces of rats throughout the building, but no dead ones were found nor were any infected ones obtained. On the other side of the street, directly opposite these premises, infected rats have been recently obtained.

Case No. 6.—Worked in a produce store in the Valley, and lived over the shop. Numerous traces of rats were observed throughout the building.

Case No. 7.—Lived at Spring Hill, and worked in the Government Printing Office. Spring Hill area supplied several cases during 1901 and 1902.

Case No. 8.—Lived opposite to the flourmill which supplied Case No. 4.

Case No. 9.—Lived in George street. Three days before she was taken ill the floors of two bedrooms on the ground floor, adjoining the kitchen where she worked, were taken up and five decomposed rats were found. These premises are adjoining some Roma-street produce stores, which about that time yielded several infected rats.

Case No. 10.—This case is discussed elsewhere. A member of the rat corps.

Case No. 11.—Lived with his parents at Downfall Creek, a scattered settlement about 7 miles from Brisbane. His father was slaughter-man at a neighbouring slaughter-yard, and deceased was in the habit of going every morning to the yard with his father's breakfast and remaining there for half an hour or an hour.

On inquiry at the slaughter-yard, the following facts were elicited:—

> Rats, until about three weeks previously, had been very numerous at the yard, but since that they had been dying in great numbers; every morning numerous dead ones were raked up and burnt; sick ones, which appeared dazed and could be easily knocked over, were frequently observed.
>
> The slaughter-yard was thoroughly overhauled, and the decomposed remains of 250 rats were obtained and sixty living ones; of these latter two were found, on bacteriological examination, to be plague-infected.

It is interesting to note that, on several occasions previous to this, infected rats were found at the head town shop of this slaughter-yard and in the immediate vicinity of this shop.

From this town shop various butchering refuse and offal were brought to the yard to be dealt with, and also the forage for the horses at the yard was first sent to the town shop from the produce stores, and then transported from the shop to the yard. It is quite conceivable that an infected rat was carried amongst the offal or the forage from the town shop to the slaughter-yard at Downfall Creek, thus causing an epizöotic amongst the rats there.

Case No. 12.—Lived at Newstead, and was occupied with domestic duties at home. The etiology of this case is obscure as no signs of rats were observed in her home, nor were any infected rats obtained in the neighbourhood, though it was thoroughly worked for rats.

Case No. 13.—Sanitary cart driver, lived at South Brisbane; his stables were on the North Quay, not far from the flourmill which supplied Case No. 4. There were numerous traces of rats at the stables, and six live ones were captured there, but they were healthy. Two carcases (probably plague-infected) were obtained, but were unfit for examination.

Case No. 14.—Lived at New Farm, and worked at a boot factory in Edward street. No signs of rats were noticed at home, but there were numerous traces of them at the factory, and on overhauling it four decomposed rats were found there. Infected rats have frequently been obtained in this neighbourhood. Case No. 3 worked in premises opposite the factory.—(*Vide also* Case No. 1.)

Case No. 15.—Lived at Woolloongabba, and worked in a brick and cement warehouse in Creek street. No signs of rats at home, but there were numerous traces throughout the warehouse. The warehouse is close to wharves which have supplied several infected rats.

Cases Nos. 16 and 17.—Lived at Billet street, Spring Hill. Case 16 was in the habit every morning of carrying breakfast to Case No. 17, at the latter's stables in Tank street; Case No. 17, who lived at Isaac street, Spring Hill, being employed as a carter. Case No. 16 usually remained at the stables for about half an hour to an hour. The onset of the disease in both these cases was practically simultaneous. About a week previous a *dazed* rat was seen in the stable-yard, and on the same day a *dead* one was also seen there.

At the stables numerous traces of rats were found, and four decomposed rats were obtained.

Case No. 18.—Living in Queen street. Traces of rats were found in some outbuildings in the yard of her home. These premises are in the immediate vicinity of shops where infected rats have been several times found during this year.

Case No. 19.—Chinese fruiterer, living in his shop in Brunswick street. Traces of rats throughout the premises, and decomposed (probably plague-infected) carcases were found.

APPENDIX A.

METROPOLITAN AREA.

TABLE I.—Showing Incidence of Plague-Infected Rats, 1900-1907.

Premises.	1900.	1901.	1902.	1903.	1904.	1905.	1906.	1907.	Totals.
Advertising Agents	...	...	...	...	1	...	...	...	1
Arcade	...	...	...	...	1	...	...	...	1
Auctioneer	...	...	1	...	...	2	...	...	3
Bacon Store	...	...	...	...	1	...	4	...	5
Bag and Bale Stores	...	...	...	...	1	1	...	...	2
Bakers	...	...	1	...	2	4	...	...	7
Blacksmiths	...	1	...	...	2	...	...	...	3
Boarding-houses	1	...	...	2	4	5	1	2	15
Bootmakers	...	2	3	1	2	...	...	2	10
Butchers	7	15	7	3	10	...	1	...	43
Butter Factories	...	..	...	...	1	...	...	...	1
Cafés	4	2	12	5	16	3	1	1	44
Carpenters	...	...	...	1	1	1	...	...	3
Chemical Works, &c.	...	...	...	...	1	...	1	...	2
Chemists	...	...	...	1	1	1	...	...	3
Chinese Dealers	...	...	...	1	4	...	...	...	5
Coachbuilders	...	...	...	...	4	...	...	...	4
Confectioners	...	...	...	...	4	...	...	...	4
Dentists	...	...	1	...	1	...	...	...	2
Drapers, &c.	3	2	...	...	1	3	1	...	10
Factories	3	3	4	...	3	...	1	...	14
Flour Mills	...	...	...	...	1	1	...	...	2
Fruit and Oyster Saloons	...	4	1	8	7	8	2	...	30
Farmers	...	...	...	...	5	...	...	...	5
Fish Dealers	...	1	...	...	2	2	...	...	5
Government Buildings	1	...	...	2	5	...	...	2	10
Grocers	8	5	4	5	25	20	4	2	73
Hotels	8	5	13	5	19	23	4	3	80
Ironmongers	...	...	1	1	...	...	...	...	2
Machinery Stores	...	...	...	2	2	3	...	...	7
Meatworks	...	...	1	...	...	...	...	...	1
Municipal Baths	...	...	...	...	3	...	...	...	3
Municipal Markets	...	...	2	...	12	2	...	...	16
Offices	1	4	3	3	8	1	5	2	27
Photographers	..	1	1	...	...	...	1	...	3
Printers	...	...	...	1	2	2	...	...	5
Produce Stores	...	2	6	1	130	11	6	...	156
Public Places	21	16	14	2	2	1	...	...	56
Railway Premises	...	...	1	...	6	...	...	...	7
Residences	15	17	16	2	29	7	3	1	90
Retail Shops	...	...	2	2	3	4	1	3	15
Saddlers	1	2	2	3	6	1	...	...	15
Schools	1	...	...	...	2	...	...	1	4
Second-hand Stores	...	...	...	...	10	...	...	...	10
Slaughter-yards	...	...	...	2	7	3	...	...	12
Stables	1	5	2	2	3	7	1	...	21
Undertakers	...	...	...	1	2	...	...	...	3
Unknown	3	3	...	...	...	...	...	...	6
Upholsterers	...	...	...	...	1	...	...	...	1
Wharves and Shipping	9	10	6	14	21	8	7	3	78
Warehouses	2	1	2	12	2	4	4	...	27
Wood Depôts	1	...	...	...	1	...	...	...	2
Total	90	101	106	82	377	128	48	22	954
Infected Mice.									
Factories	...	...	...	1	...	...	...	...	...
Grocers	...	...	...	...	...	1	...	...	...
Produce Stores	...	...	...	...	2	...	...	...	...
Residences	...	...	...	...	1	...	...	...	...
Warehouses	...	...	...	1	...	...	...	...	...
Total Mice	...	...	...	2	3	1	...	...	...
Totals of Rodents	90	10	106	84	380	129	48	22	960

APPENDIX B.

TABLE II.—Showing Occupation Incidence (Whole State, 1900-1907).

Occupation of Persons Attacked.	Male.	Female.	Total.
Food Trades and Trades Associated with Rats—			
Bakers	4	...	4
Butchers and Slaughterers	12	...	12
Confectioners, Cooks, Waiters, Stewards, &c.	13	6	19
Fishmongers	2	...	2
Fruiterers	5	2	7
Grocers and Provision Stores	14	6	20
Grooms, Jockeys, Carters, &c.	8	...	8
Hotels, Restaurants, &c.	21	5	26
Produce Stores	26	3	29
Ratcatchers and Scavengers	8	...	8
Wharf Labourers	17	...	17
Warehouses, Packers, &c.	13	1	14
			— 166
Other Trades—			
Bootmakers	5	...	5
Bookbinders, Printers, &c.	1	...	1
Blacksmiths	2	...	2
Carpenters	2	...	2
Drapers, Tailors, and Shop Assistants	8	2	10
Ironmongers	1	...	1
Painters	2	1	3
Plumbers	3	...	3
Saddlers	3	...	3
Timber Yards	4	...	4
			— 34
Housework—			
Domestic Duties (including Servants, and Children at home)	3	61	64
School Children	46	18	64
			— 128
Clerical Work	10	2	12
Medical Practitioners, Nurses, Wardsmen, &c.	6	5	11
			— 23
Chinese	22	...	22
Kanakas	2	...	2
Miscellaneous Occupations (including Cane-cutters)	40	10	50
No Occupation	30	9	39
			— 113
Total	...	...	464

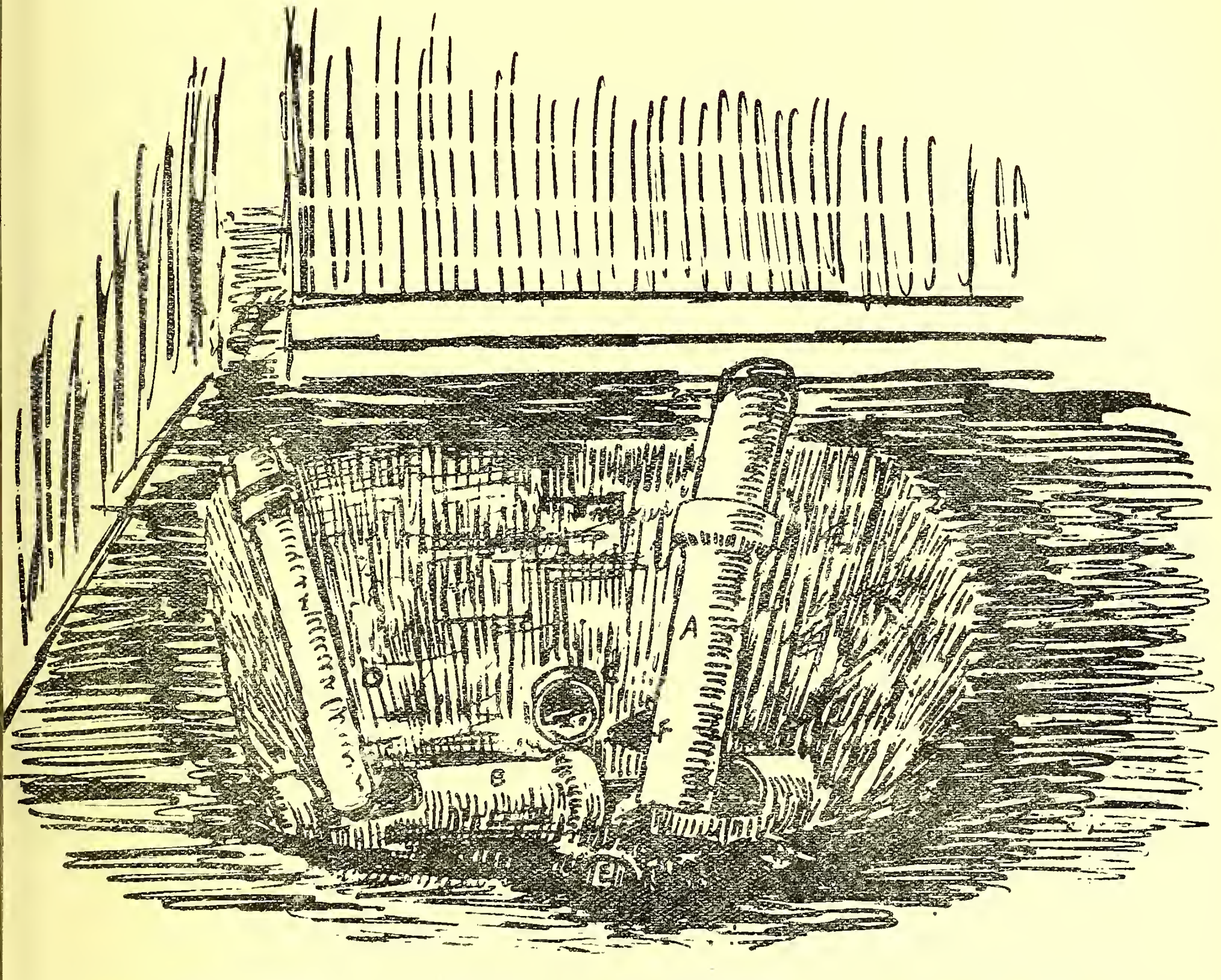
APPENDIX C.

TABLE III.—Showing Incidence on Households and Secondary Cases.

Eleven Premises (dwellings or places of emyloyment) at which Twenty-five Cases received their Infection.

Year.	Initials.	Age.	Sex.	Occupation.	Place where Secondary Case Occurred.	Date of Attack.	Remarks.
1900	M. C., junr.	20	M.	Barman, Hotel	...	25 July	Recovered (son).
	M. C., senr.	...	M.	Hotel proprietor	Hotel, Leichhardt street	26 ,,	ditto (father).
1901	J. H.	18	M.	Soldier	...	5 April	Recovered (son).
	K. H.	44	F.	Housewife	Mary street, Rosalie	6 ,,	ditto (mother).
	W. E. W.	18	M.	Bootmaker, Leichhardt street	...	17 May	Died (brother).
	G. W.	21	M.	Ditto	Leichhardt street	17 ,,	ditto ditto.
1902	H. L.	19	M.	Dentist's assistant, George street	...	19 February	Recovered.
	J. C.	23	M.	Ditto	George street	27 ,,	ditto.
	W. W.	37	M.	Commission agent, Tank street	...	10 March	Died (father).
	M. E. W.	...	F.	Housewife	Tank street	10 ,,	Recovered (mother).
	H. W.	16	M.	Schoolboy	ditto	10 ,,	ditto (son)
	G. S. B. H.	44	M.	Commercial traveller, Peel street, South Brisbane	...	12 ,,	ditto
	A. B.	20	F.	Domestic servant	Peel street, South Brisbane	14 ,,	ditto.
	J. B.	27	M.	Barman, Hotel	...	26 ...	Died.
	S. C.	23	F.	Housemaid	Hotel, George street	30 ,,	ditto.
	E. L.	40	F.	Housewife, Railway Reserve	...	27 April	Recovered (mother).
	G. L.	14	M.	Schoolboy	Railway Reserve	27 ,,	ditto (son).
1904	M. R.	32	F.	Housewife, Melbourne street	...	19 August	Recovered (mother).
	V. R.	12	F.	Schoolgirl	Melbourne street	20 ,,	ditto (daughter).
1907	M. S.	23	F.	Housewife, Duncan street	...	7 April	Died (mother of baby).
	M. S.	15	F.	At home	Wandoo street	9 ,,	Died (sister of above).
	Baby, S.	1 week	M.	Born at Lady Bowen Hospital, ta	ken to Plague Hospital	9 ,,	Died.
	R. R.	18	M.	Yardman, Hotel	...	24 May	Recovered.
	G. J. W.	17	M.	Ditto ditto	Hotel, Queen street	27 ,,	ditto.
	J. C.	45	M.	Proprietor, ditto	...	26 ,,	ditto.

APPENDIX D.



PEN AND INK SKETCH OF BRISBANE DRAINAGE FOUND IN GEORGE STREET IN OPENING UP RAT BURROWS, 17th FEBRUARY, 1905.

A.—A 6-inch pipe recently laid.
B.—A 6-inch pipe evidently a sectional drain.
C.—A 4-inch pipe discharging anywhere.
D.—A 4-inch pipe from rain-water pipe.
E.—A 4-inch pipe discharging anywhere.
F.—A hole through which sewage comes; pipe not in sight.

APPENDIX E.

REPORT ON THE OUTBREAK OF PLAGUE AT MARYBOROUGH.

May-June, 1905.

Department of Public Health, Queensland,
Brisbane, 29th June, 1905.

To the Hon. the Home Secretary.

Sir,—I have the honour to submit the following Report on the recent Outbreak of Plague at Maryborough, in this State.

On the 14th June instant, an official inquiry was held by me at the Municipal Chambers, Maryborough, into the circumstances of the outbreak of the disease in that area, a city of 12,000 inhabitants, situated on the banks of the Mary River, about 20 miles from its entrance, and distant 167 miles by railway from Brisbane.

The outbreak was characterised by pneumonic manifestations, without distinct enlargement of the lymphatic glands—a type of plague which, fortunately, has been rare in Queensland. The members of a family, named O'Connell, consisting of seven children, residing with their father at Newtown, near the city, in a state of great poverty and neglect—father out of employment, and of intemperate habits; mother dead—were seized within a few days of each other with acute gastro-intestinal symptoms, which terminated in five of the cases with symptoms of pneumonia. Of nine persons attacked (five in one family), eight died within a short period, including two nurses who were attached to the local General Hospital staff.

The first case (John O'C.), a boy, aged seventeen years, sickened on the 19th May, and lay ill at his home for six days prior to the visit of a local medical practitioner a day before death on the 25th May. No suspicion of the nature of the illness was aroused, and the cause of death was certified as "pneumonia." This boy was employed as an assistant in a fruiterer's shop situated in the main thoroughfare of the town. An open sewer—a natural watercourse—receiving the main drainage of the city, runs beneath and alongside a block of shops, including the fruiterer's shop referred to, and affords an excellent harbourage for rats.

On the 28th May, a neighbour of the O'Connell family (Mrs. E.), aged forty-nine years, who had visited and assisted in nursing the boy John O'C. before his death, sickened with headache, vomiting, severe pain in the epigastrium and back, and died at her home on the 31st May. In this instance a certificate of death was refused by the local medical practitioner called to attend the case pending a *post mortem* examination of the body by the Health Officer.

Meanwhile, four other children of the O'Connell family had sickened with headache, pyrexia, severe vomiting, pains in the cardiac and epigastric regions, and were removed to the General Hospital on the 28th May. On the 31st May, a girl (Ellen O'C.), aged seven years, and a boy (James O'C.), aged fifteen, died at the hospital with symptoms of "broncho-pneumonia." Two other members of the family —the eldest girl (Kate), aged eighteen years, and a younger sister (May), aged nine years—both recovered, and were discharged as convalescent on 6th June. They were removed as "contacts" to the Plague Hospital, and have since been discharged.

On the 28th May, two further members of the O'Connell family—a boy (Ritchie), aged ten years, and a girl (Mary), aged three and a-half years—were taken to the home of a girl friend (Miss S.) of the family, and nursed by her till the next day, when they were admitted to the General Hospital on the application of the father, Richard O'Connell.

Ritchie died on the 2nd June, and Mary on 3rd June, at the General Hospital, both with symptoms of plague-pneumonia.

Importance now attaches to the new centre of infection—the wards of the General Hospital—and the association of the nurses with, at the time, unrecognised cases of plague with more or less marked pulmonary symptoms.

Nurse B., aged twenty-two years, a staff nurse on night duty, had charge of the O'C. family from their admission until 30th May. She was taken ill on 3rd June with chest symptoms, cough, and expectoration, and died on 6th June.

Nurse W., aged twenty-eight years, another staff nurse, who also attended the O'C. cases, as well as Nurse B. referred to, presented clinical symptoms of lobular pneumonia on 5th June, with cough and watery sputum. She died on 12th June.

A third nurse, E. S., aged twenty-eight years, who attended Nurse W., sickened on 10th June with headache, pyrexia, and general malaise. She recovered, and was discharged convalescent on the 23rd June.

The above is a short history of the association and distribution of the cases. It will be seen that a direct association is established between all of them, which connection is also greatly strengthened by the time incidence of the attacks.

A study of the symptomatology and type of the disease emphasises the fact that plague may manifest itself in a form other than the most frequent and most typical—*i.e.*, bubonic plague, which up to the present time has characterised every outbreak in the State.

In all of the above cases the variety of plague partook of the nature of a general infection rather than of a local bubonic character. In no case was there any distinct enlargement of the lymphatic glands accessible for examination.

All the O'Connell cases were probably septicæmic at the outset, and received infection from the same source. With the exception of the first case (John), who died at home after an illness of seven days, pulmonary symptoms did not develop until after their admission to the General Hospital.

A careful search for rats at the time in the vicinity of the sewer abovementioned elicited no evidence of plague infection among the rodents, which, however, were found in large numbers, many of them being of exceptional bodily size and vigour.

The squalid surroundings of the O'Connell family—the majority of the family were practically dependent upon the charity of neighbours—their ill-nourished bodies, and insanitary dwelling abode (the

Municipal Inspector reported that the interior of the house was in a filthy state), were doubtless predisposing causes in lowering individual vitality and the natural resistance to infection.

The contact (Mrs. E.) who attended the first case (John O'C.) just prior to his death, and at a time when cough and secondary pneumonia had deevloped as a manifestation of septicæmic plague, evidently contracted primary plague-pneumonia by "drop-infection," the atmosphere immediately surrounding the patient containing minute suspended particles of expectoration.

The young woman S., who was also a "contact," did not attend the first O'Connell case (John) until after his death, when she assisted in "laying out the body." The circumstance that she escaped infection, although in close and constant attendance on the other affected members of the family, may be attributed to the fact that pulmonary symptoms had not at that time developed in the sick children. There is no evidence that the indisposition of either the eldest girl, Kate, or her younger sister, May, the two surviving members of the O'Connell family, was due to plague.

Special interest attaches to the cases among the hospital nursing staff as affording an illustration of a variation both in the type of the disease and severity of attack. As in the unfortunate case of Mrs. E., the two hospital nurses who succumbed to the disease obtained their infection by direct contact with septicæmic plague cases, which subsequently developed secondary pneumonic symptoms. The illness of Nurse B., as well as Nurse W., was primary pneumonic in type, the development of the disease being due to the growth of the plague bacilli in the lung tissue.

In the case of Nurse S., who is now convalescent, pneumonic symptoms did not develop, her symptoms of more or less serious indisposition being modified, doubtless, by an initial immunising and subsequent repeated doses of Yersin's serum.

The measures taken for the suppression of the outbreak were preventive inoculation of doctors and nurses in attendance, and the wearing of specially designed costumes and respirators; isolation of the infected; segregation of the probably infected (quarantine of the hospital premises); destruction by burning of O'Connell's house with its contents; disinfection of clothes; disinfection of houses, including the General Hospital; cleansing operations, and the destruction and examination of rats. It is to be regretted that the precautionary measures taken by the Department of Public Health, and which were carried out with success after the death of the seventh patient, could not have been enforced at an earlier date.

Apparently, there was nothing to arouse suspicion until the concurrent deaths of Mrs. E., at her residence, and the two children in the General Hospital the same day—viz., 31st May. The local practitioner who attended the first case in the O'Connell family certified the cause of death to have been one of ordinary pneumonia. On being called in a few days after to see several other sick members of the family, he ordered their removal to the General Hospital. The nurses and inmates were thus exposed to infection before the real nature of the disease had been discovered. There was a rapid development. Of the six deaths which occurred at the hospital, five took place within six days. It was only after several sudden deaths, arising from some suspicious but undetermined cause, had occurred that the Department was communicated with.

On the 31st May, Dr. Lee Garde, the medical superintendent of the hospital, and Dr. Dixon, a local practitioner who had attended Mrs. E., reported the O'Connell cases to the Mayor as suspicious, and probably plague. His Worship communicated the report to me. The same day, Dr. Penny, the Government Medical Officer at Maryborough, telegraphed to me at Brisbane as follows:—

> "Boy and girl died at hospital. Woman who attended also died, all of broncho-pneumonia, after three days' illness. Two sisters and one boy still ill in hospital. Suspicious. Want *post mortem* examination on woman."

I telegraphed the following reply:—

> "Take all precautions. Isolate cases and contacts as far as possible. Send 'specimens' of organs from *post mortem* case here for examination. Will send my Health Officer and serum if symptoms still suspicious. Wire me progress to-day."

The same day (1st June) I received the following reply:—

> "Precautions taken. Serum injected; *post mortem* examination refused. Body buried without certificate. Think plague. Circumstances suspicious, not conclusive. No glandular enlargement. Patient same. Sputum sent 'Pound' (Government Bacteriologist)."

On 2nd June the Government Bacteriologist furnished me with the following report:—

> "Four smear preparations received from Maryborough this morning. The specimens were unsatisfactory. In one I found a few organisms which were apparently pneumo-bacteria. No *Bac. pestis* were detected."

Under the Plague Regulations of 1903 all cases of plague, or suspected plague, must be immediately reported to the Health Officer of the Local Authority, and to the Commissioner of Public Health.

When plague threatens, the recognition of the first case or cases is of paramount importance. In times outside those of an epidemic, plague diagnosis is more or less a matter of probability, the disease not unfrequently being mistaken for typhus, malaria, pneumonia, and other diseases. The pneumonic type, particularly, is likely to be overlooked. On the 11th February, 1901, a circular letter was sent by me to all the Medical Officers of Health and Government Medical Officers throughout the State, especial attention being directed to the following paragraph:—

> "Any sudden death occurring during a plague epidemic, or threatened epidemic, should be regarded as suspicious, and a *post mortem* examination made."

Had the *post mortem* examination on the body of Mrs. E. been carried out as directed by me on the request of the Government Medical Officer, on 31st May, there is every reason to suppose that the real nature of the disease would have ben brought to light. Precautionary measures could have been taken which would have materially lessened the danger and risk to the doctors and the nurses attendant on the cases at the hospital.

The adoption of compulsory notification should be combined with an efficient system of registration of deaths.

> "No scheme of death registration can be perfect unless it furnishes information in respect of the *causes* of deaths as well as of the numbers."

In the absence of reliable death certificate this information can only be obtained by *post mortem* examination, or an inspection of and inquest on the body.

The burial of the body of Mrs. E. without a death or other certificate by the Maryborough Cemetery authorities was strongly commented on by me at the official inquiry. Regulation 19 of the "Rules and Regulations made by the trustees of the Maryborough Cemetery pursuant to '*The Cemetery Act of* 1865,' and published in the *Government Gazette* of 8th November, 1870," reads:—

> "No funeral will be allowed to take place (except under circumstances of the most urgent nature) without a certificate of the registration of the death, signed by the district registrar, or a certificate of a coroner or magistrate holding an inquest or inquiry; such certificate to be delivered to the sexton *before* the funeral enters the gate of the cemetery."

Harry Nicholas Hansen, sexton at the cemetery, in his evidence stated:—

> "If I do not receive the certificate the undertaker generally tells me 'it is all right.' I cannot very well stop a whole congregation outside the gate if there is no certificate. I have let in many funerals without a certificate a score of times; it has often been done in the past, even before my time. I have taken the word of the undertaker that everything was right. I never reported these cases to the cemetery trustees, it having been a practice. Have been employed for twenty-seven years, and have been sexton for twenty-three years. I admit it was not right to bury without a certificate, but will take good care it does not happen again."

Frederick Vaughan, Police Magistrate at Maryborough, said:—

> "All deaths under suspicious circumstances should be reported to the police, and, if required, I give an order for a *post mortem*, which the Government Medical Officer carries out and reports upon. If further inquiry is required, I hold it; when a doctor refuses a death certificate, an order for a *post mortem* is given. The police would report such refusal to me if it came to their knowledge. This is the first I have heard of the case, and I have no knowledge of it. Someone has committed an illegal act, and I am surprised to hear of it. I should think the person responsible would be the man who allowed the body to be taken into the cemetery, as the proper procedure was for the certificate to have been produced at the cemetery gate."

The fact that a death certificate had been refused, the suspicious circumstances surrounding the case justifying the medical attendant in the action he took, or that a *post mortem* examination had been objected to by the husband or relatives of the deceased, should have been reported to the police magistrate. No such report was made.

On receipt of my telegram the Health Officer proceeded to make the examination, and found that the body had already been removed and buried.

On the 2nd June, Dr. Baxter-Tyrie, Health Officer to the Department of Public Health, left for Maryborough to investigate the nature of the illnesses. On the evening of that day he assisted the Government Medical Officer at a *post mortem* examination on the body of Richard (or Ritchie) O'Connell, at the hospital morgue. The upper two-thirds of the left lung was dark, solid, red hepatisation being well marked. Sero-sanguineous fluid exuded from cut surfaces. Section sank in water. Several hæmorrhagic condensations about size of a shilling over middle lobe of left lung. Sero-sanguineous fluid exuded from mouth and nostril, and filled bronchial tubes. Liver large, tough, and intensely hyperæmic. No glandular enlargement. Bacteriological examination of sections of lungs, liver, spleen, kidneys, and heart sent to the Government Bacteriologist showed numerous *Bac. pestis.*

A guinea-pig inoculated from above material died in four days. *Post mortem* examination revealed the characteristic appearances of plague, and microscopical preparations from the spleen showed numerous typical *Bac. pestis.*

Dr. Tyrie found a large number of *Bac. pestis* in the sputum which had been collected before death.

The sections of viscera above referred to were despatched from Maryborough on the evening of the 2nd June, and should have reached Brisbane early next morning, the 3rd instant. They were not received, however, at the Bacteriological Institute, Brisbane, until the 5th instant—a delay of two days.

Stephen O'Brien, post and telegraph master at Maryborough, stated that the delay occurred at Brisbane. He furnished a copy of the inland parcel post bill to show that the parcel containing the "specimens" had been despatched from the Maryborough Post Office on the night of the 2nd June, and a receipt for the same from the receiving officer at Brisbane on the 3rd instant.

On making inquiries at the Brisbane office on the morning of the 3rd, the postal authorities stated that no packet had been received from Maryborough. I am satisfied that the Maryborough officers are in no way to blame for the delay. A mistake appears to have been made in despatching the "specimens" by parcel post instead of by packet in the ordinary manner. The fact that a Saturday and Sunday intervened may also have delayed the delivery of the parcel until the Monday (5th instant).

On the 3rd June the local fire brigade burned down the house lately occupied by the O'Connell family. Everything the house contained, with the exception of a few articles of hardware and crockery, were burned with the house. The mattress upon which John O'Connell slept had been burned at the time of his death.

The house occupied by Mrs. E. was also thoroughly fumigated, and the clothing and bedding of the unfortunate woman burned or disinfected.

In company with the members of the Health Committee of the Municipal Council, Dr. Tyrie inspected a number of places in the centre of the town, including butchers' shops, produce stores, &c. One large produce store and several other buildings were condemned, and ordered to be pulled down.

A gang of men employed by the Municipal Council for the purpose of cleansing and rat-destruction were placed under the superintendence of Chief Inspector Simpson, of the Health Department.

On the 3rd June a *post mortem* examination on the body of Mary O'Connell was conducted at the hospital morgue. The lower lobe of the left lung was found to be in a state of red hepatisation with recent pleurisy over the affected area. No glandular enlargements; *Bac. pestis* found in lung tissue. Bacterioscopic examination of the sputum before death revealed the presence of *Bac. pestis*, pneumococci, and streptococci.

The thoracic organs were removed *en masse*, and placed in formalin for future examination.

On 5th June, Dr. Tyrie informed me by wire that a nurse attending the O'Connell cases had developed pneumonia, and was seriously ill. Two departmental nurses, experienced in plague, were sent by me to relieve the nursing staff of the Maryborough Hospital. The death of Nurse B. was reported on 6th June. The sputum from this case contained *Bac. pestis*, pneumococci, and streptococci. On the morning of 8th June another nurse of the General Hospital staff was reported to be seriously ill, with symptoms of lobular pneumonia. The sputum contained enormous numbers of *Bac. pestis*, and she died on 12th June.

Two aditional nurses of the Health Department plague staff were sent to Maryborough to attend the affected nurse of the local staff.

On 7th June, Dr. Love, Hon. Pathologist to the Department, proceeded to Maryborough at my request, to confer with Dr. Tyrie, and to carry out certain investigations.

With reference to the case of Nurse W., Dr. Love, in his report of the 11th June, states—

> "The case was, of course, isolated from the first, but there was unnecessary communication with the sickroom by the other nurses and matron. No respirators or overalls were being worn, the net of the bed was not drawn, and no sheets or screens soaked in disinfectant used. The nurses of the Health Department staff soon put these matters in order, but as the other nurses had been unduly exposed to infection, I ordered all of them, including the medical superintendent, the matron, and the wardsman, to have 20 c.c. of serum injected, and a dose of 10 c.c. to be administered to the nurses attending on the cases each morning till the period of incubation was over (five days). One nurse (Nurse S., who subsequently was taken ill and recovered) objected, and was still allowed to nurse the case, but on being told by me that she could not be allowed to take the risk without inoculation, she consented, and was inoculated some twenty-four hours later than the others. The nurses were instructed in the precautions to be observed. All secretions were promptly disinfected. The conclusion I formed of this case was that it was one of primary pneumonic plague. There were no glandular enlargements."

The hospital premises were strictly "quarantined," and the local medical and nursing staff forbidden entry to the infected ward, which was strictly isolated and placed in charge of the departmental health officer and nursing staff. Part of the clothing and bedding of the nurses who had died were burned, and the rest disinfected.

On 12th June I proceeded to Maryborough to inquire into the circumstances of the outbreak, and to satisfy myself that every precautionary measure had been adopted. Urgent departmental duties, demanding my attendance at Brisbane, prevented an official visit at an earlier date.

The matron of the Colmslie (Brisbane) Plague Hospital was placed in charge of the ward where the remaining case (Nurse S.) was isolated, and Dr. Tyrie, the Health Officer to the Department, was given sole control of this and any further cases likely to develop.

All the medical officers and nurses coming in contact with the patient received an immunising dose of 20 c.c. of Yersin's serum. Specially designed overalls and respirators were worn at the bedside or in the ward.

The quarantine period of the inmates of the hospital, who were all segregated in the east wing of the building, was strictly observed.

Three of the O'Connell cases and Nurse B. had been located in the east wing. The General Hospital was released from quarantine on 16th June. The more urgent cases were removed in the ambulance wagon to the Reception House, which had been fitted up as a temporary hospital, the rest of the inmates, being convalescent, going to their homes or to friends.

Nurse S. was discharged convalescent from the General Hospital on 23rd June.

No further cases have been reported, and there has been no further development.

All the wards of the hospital, nurses' quarters, &c., have been thoroughly disinfected under the superintendence of officers of the Health Department.

The local rat-catching gang has been supplemented by five experienced men from the gang employed by the Health Department. Of 221 rats and 45 mice examined, only 2 rats have been found plague-infected.

The cleansing and rat-catching operations are still proceeding.

The town now presents a cleanly appearance, which reflects the greatest credit upon the householders and the Local Authority. His Worship the Mayor and the Municipal Council afforded the officers of the Health Department active and willing co-operation.

I wish to express my appreciation of the valuable service rendered by Dr. J. A. Cairns Penny, the Government Medical and Health Officer at Maryborough.

I have the honour to be,

Sir,

Your obedient servant,

B. BURNETT HAM, M.D.,
Commissioner of Public Health.

PART III.

C.—THE MEDICAL ASPECTS OF PLAGUE.

I.—CLINICAL FEATURES.

In most of its clinical characters and pathological features, plague in Queensland did not differ to any material degree from the recorded descriptions of the disease by observers in other parts of the world. Since a general description of the clinical symptoms and the principal types are to be found in the current and already voluminous literature on plague, no useful purpose could be served here by a repetition in detail of the clinical varieties or types of plague. So many writers have prefaced their remarks on the clinical features of plague to the same effect, that the only excuse for a similar apology lies in the fact that many detailed descriptions of the clinical types of plague mean little more to the general practitioner than a bewildering list of symptoms that may, or may not, be present, in the many symptomatic manifestations of the recognised types of the disease.

It may, therefore, prove more profitable if I endeavour to summarise such information as I have been able to collect concerning the fixed clinical types generally, and the more conspicuous features exhibited by individual cases in particular, in the series of epidemics which came under our observation.

Classification.—There are three well-defined types of plague—the *Bubonic*, including those milder forms known as ambulant or abortive plague or *Pestis minor*; the *Septicæmic*, including intestinal and fulminant plague, and cases with secondary pneumonia; and the *Primary plague pneumonia*, or *Pestis pneumonica*, characterised by early appearances of lung symptoms and a high mortality.

Whatever basis be adopted for the purpose of convenient classification of the various types of plague, it is obvious that there is but one essential or real cause " which indicates its oneness, under whatever symptomatic modifications it appears."

The classification of the older writers is one based rather on distinction than of degree, the diversity or severity of the symptoms, and especially the prominence of one or several of them, leading to the numerous types of plague which have been more or less defined.

Cases have occurred in the Queensland epidemics which, on the one hand, were characterised by an adenitis in one or several lymphatic glands, with little constitutional disturbance; and on the other hand, by a virulent and general septicæmia, without primary buboes, which quickly culminated in the death of the patient. The history of the Maryborough epidemic in 1905 afforded an illustration of variation in the usual type of the disease and the severity of attack which, fortunately, was not often encountered.

While, in most of our cases, there was a more or less strict adherence to type, there was also a great diversity in the symptoms of individual cases. In the simple bubonic type, which mainly was the form met with in our cases, experience taught us that infectivity, in the sense of being communicable by direct infection from man to man, was practically nil; but that in the primary pneumonic cases, or where secondary pneumonic had supervened in the course of a septicæmic case, we had to deal with a disease which was directly and readily transmissible by human agency.

The Bubonic Type.—The clinical picture presented by a patient suffering from plague in its most usual form may be described as follows:—When seen by the health officer, or medical attendant, at an early stage of the disease, the patient is usually found in bed with one leg flexed at the hip, or one arm supported by the bed clothes, indicating an effort to lessen pressure upon a bubo in the groin or axilla. The expression is one of anxiety, with suffused eyes and flushed face. The skin is hot and dry, and the respirations increased. The temperature varies from 102° F. to 105° F., and at this stage it is invariably high. The pulse is quick, full, soft, and dicrotic. The tongue is moist and coated with a thick white fur along the dorsum, the tip and edges being clean and of a bright pink colour. The bowels are constipated, and there is often intense thirst. In many cases the speech is husky and slow, questions being answered in an irritable tone, as if the patient was annoyed at being disturbed. Delirium is not often observed at this early stage; disturbance of the cerebral functions not frequently manifesting itself until after the patient had been removed to hospital. The want of sleep is commonly complained of, and the patient generally presents the aspect of one stricken with an illness of some severity. A slight but distinctive odour is often perceived about the patient. The distinguishing feature of the disease is the bubo. It commonly makes its appearance on the second day of illness, the pain and tenderness of the swelling soon attracting the patient's attention to its presence. The primary bubo is usually femoral, one of the upper external or internal of the femoral group of glands being chiefly affected. Femoral adenitis, with a soft and dicrotic pulse, are symptoms very characteristic of plague. The history generally given is that the patient was in good health until the day before, when he was suddenly taken ill with fever, intense headache, vertigo, pains in the back and limbs, nausea, vomiting, and a rigor; or in the case of young children, with convulsions.

No definite history of prodromal symptoms can be obtained, and, in our experience, prodromata were extremely rare. The onset of illness was distinctly sudden.

The illness having attained the degree of severity above described during the first forty-eight hours, the constitutional symptoms now increase in degree, reaching their maximum about the third day and lasting usually to the fifth or sixth day. The patient now lies in a feeble and listless condition suggestive of extreme nervous and muscular prostration. The temperature has fallen a degree or two, but the pulse has increased in rate; become weaker, smaller, irregular, and more dicrotous. The tongue is still covered with fur, which is now of a brown colour, the tip and edges of the tongue being dry and of a deep red or mahogany colour. The patient lies totally indifferent to his surroundings, muttering and talking to himself, or occasionally uttering noisy outcries. When roused to answer a question, there is often a moment or two of returning consciousness, the answer frequently being given more or less correctly but in a blurred syllabic or husky tone, like that of a drunken man. Often delirium is followed by lethargy and coma in the more severe cases. An examination of the bubo shows that it has rapidly increased in size, with marked periadenitis and an inflammatory blush on the skin over it. Its ultimate size varies considerably. Albumen is found in the urine, generally in small quantity, and associated with hyaline and epithelial casts. In favourable cases, the constitutional symptoms begin to abate about the sixth to the tenth day; the temperature falling to normal, the face losing its congested appearance, the tongue clearing, and the delirium subsiding. Sleep at this stage usually returns, the patient often appearing very lethargic. The pulse is still feeble and easily compressible. The bubo continues to enlarge, but now begins to soften. About the tenth day the stage of convalescence is reached, the skin becoming moist, the pulse slower and stronger, and altogether the patient wears a brighter aspect. With the fall of temperature pain and tenderness usually depart from the bubo which rapidly proceeds to the suppurative stage, the abscess being commonly opened about the end of the second week. Complete restoration to health is usually tardy, the period of convalescence being often protracted by a continued discharge from the bubo. The average stay in hospital is about thirty days.

When, however, the termination is not by recovery, death is more frequently caused by sudden heart failure about the third or fourth day.

Sudden failure of the heart's action is a grave possibility in all stages of the disease up to and sometimes including the period of convalescence.

Buboes.—Swelling of one or more of the external lymphatic glands was exhibited in about 75 to 80 per cent. of our cases. As a rule the primary bubo appears within the first few days, most commonly the second day; in a few cases its appearance was delayed although constitutional symptoms of plague were present from the first. In the group of schoolboy cases of the New Farm outbreak, in January, 1907, buboes did not make their appearance in several of the cases until the third or fourth day, although the patients were severely ill and delirium was present on the second day. The late appearance of the primary bubo is of some importance in estimating the significance of the swelling as a result of a minute lesion—*e.g.*, a fleabite—in the area drained by the affected gland. It was extremely rare to find anything like an accompanying lymphangitis.

Multiple buboes were not often met with from the first, but secondary glandular swellings were not infrequently met with in other regions than the groin during the course of the illness. On its first appearance, the *primary* bubo was about the size of a marble, but rapidly increased in size to that of a horse-chestnut or a pigeon's egg. In one instance a cervical bubo attained the dimensions of a cricket ball. The skin over the affected gland was reddened at first, deepening to a purplish tint later on. In its simplest form the primary bubo consisted of one gland, most frequently in the femoro-inguinal group, and in a large proportion of cases the left femoral gland was the one chiefly involved. Not infrequently the groin buboes were associated with cervical and axillary buboes. Cases with independent cervical buboes invariably proved fatal.

Exceptionally, glandular enlargements in the supratrochlear, submaxillary, popliteal, and brachial regions were noted. The superficial or external bubonic enlargements, however, did not represent the total extent of the involvement of the lymphatic system; the deeply-situated glands, the pelvic and iliac, as well as the retro-peritoneal, and sometimes the mesenteric being likewise affected. This phenomena was often observed in autopsies on fatal bubonic cases which had become septicæmic. In the case of a femoral or inguinal bubo, the chains of glands in the pelvic and abdominal regions were frequently associated in the general swelling of the superficial glands of the groin as to form one large continuous bubo.

The progress of the primary bubo depended on the stage at which the inflammatory process was arrested. In the mildest cases, resolution took place without breach of the skin surface, but in the majority of cases the bubo went on to soften and eventually to suppurate. After opening of the abscess, the healing was often delayed by a discharging sinus which took weeks and even months to heal, necessitating the detention of the patient in hospital when convalescence had otherwise been well established.

The distribution of the buboes was as follows:—

Left femoral ...	109 cases.	Right cervical ...	10 cases.
Right femoral ...	90 ,,	Left cervical ...	12 ,,
Left inguinal ...	50 ,,	Submaxillary ...	4 ,,
Right inguinal ...	48 ,,	Supratrochlear ...	2 ,,
Femoro-inguinal	50 ,,	Popliteal	2 ,,
Right axillary ...	16 ,,	Multiple buboes ...	6 ,,
Left axillary ...	14 ,,		

Nervous System.—Symptoms indicating derangement of the nervous system appeared early, but usually after admittance of the case to the hospital. Severe frontal headache, vertigo, pains in the back of the head, limbs, and chest, sleeplessness and restlessness were most commonly complained of in the early stage of the illness. During the febrile stage there was more or less active delirium of a low muttering type. There was often a constant desire of the patient to get out of bed. When the illness had become fully developed, delirium or coma of a varying degree, loud screaming, noisy outcries, thickness and impairment of speech, or staggering gait on attempting to walk, were frequently noticed. Acute mania and hallucination are recorded in three cases cited below. Lesions of the special senses, especially of the organs of vision, were also observed. Illustrations of the more typical cases exhibiting these symptoms are submitted in the following clinical notes:—

Bubonic Plague Complicated with Mania. Recovery.

*Case No. 11, 1901.—Aged 42, living at Red Hill, was reported by her relatives as being ill with symptoms suggestive of plague. The patient was quite well until the 5th April, 1901, when she noticed a slight swelling in her right groin, and complained of headache and vomited a little. On the 6th and 7th she felt much the same. On the 8th she became slightly delirious. On admission to the Suspect Hospital the temperature was 103.6, pulse 112. The face was flushed, eyes injected, speech thick and hesitating. The mind was wandering, and the patient had some delusions that her people were not kind to her, and were trying to work her some mischief. Her case suggested alcoholism to a remarkable degree, but this factor was safely excluded. The surface of the body was very extensively fleabitten. There was a femoral bubo about the size of a small pigeon's egg. It was not in the least tender. There was no redness of the skin over it. It was found to contain plague bacilli in enormous numbers. At 9.30 p.m. she had 100 c.c. of curative serum subcutaneously. She complained of feeling cold, but did not shiver, at 11.30 p.m. She then slept about half an hour. On waking up, the patient became very restless and delirious, and did not sleep again. At 2 p.m. the temperature registered 104 and pulse 112. Next morning the temperature was 103 and pulse 88. The mental condition had improved, and during the afternoon she was transferred to the Plague Hospital, arriving there at 3.45 p.m.

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* "Report on Plague, Brisbane, 1901-2," by Dr. A. C. F. Halford.

Condition on Admission.—Temperature 102·8, pulse 84. Quite rational, and complained of frontal headache. The face was still flushed, and eyes markedly injected. Speech thick, cerebration slow. There was some tenderness in the bubo. At 4 p.m. she had 100 c.c. of curative serum subcutaneously at side of chest. She took food well, and slept fairly well during the night. On the morning of the 10th her temperature was 101 and pulse 72. During the morning she suddenly became excited and noisy for a few minutes, and refused her food. She then became sensible and quiet, taking her nourishment kindly until about 11 p.m., when she suddenly sat up in bed and talked excitedly and loudly for a few minutes. She then quietened down, and slept till about 6 a.m., when she had another attack of delirium, and got out of bed. During the night she perspired continuously, and the temperature had fallen to 99·2 and pulse 72.

On the 11th she had a quiet day, and seemed quite sensible. She took her food well and slept well all that night.

Next day she was still quiet, and took her nourishment without difficulty. She remained awake all night and became excitable, getting out of bed, &c. In the morning (13th) she refused to speak or take her drink, and stared at the nurses with a fixed gaze. At 12.30 p.m. she jumped out of bed, exclaiming that somebody was going to murder her. She was got back to bed, and gave no further trouble that day, and slept fairly well that night. Her pulse and temperature were normal. On the 14th, quiet all the morning, will answer when spoken to, and taking food a little better. During the afternoon she absolutely refused to speak, and would take no nourishment. At 8.15 she jumped out of bed, saying, "I am going to be murdered," several times. She slept for about an hour, and then became restless, jumping out of bed and screaming. This continued for about another hour, when she went to sleep, and slept for the rest of the night.

She continued to be subject to these fits of mania for about a *fortnight*, and sometimes became very violent and required forcible restraint. She was never dangerous, however. After this the attacks became milder and less frequent, and at the end of another week she seemed to have completely recovered. After a week's further detention she was discharged to the care of her friends on the 15th of June. At her home she became discontented, and, although treated with the utmost kindness, she began to resent what she imagined to be unfeeling treatment of her relatives, whom she accused of scheming against her interests and welfare. By the end of June she became unmanageable, and for her own protection and for the sake of appropriate treatment she was certified as a *lunatic* and admitted to Goodna Asylum for the Insane. In less than two months she had completely recovered, and was once more sent to her home. There has been no return of the malady since.

Acute Delirium.

*Case No. 4, 1901.—Male, aged 6½ years. Admitted to Children's Hospital on 15th March, 1901, on the fifth day of disease. Temperature on admission 105·6, pulse 144, respirations 36. Had no serum. Prominent feature, *screaming delirium.* Death from heart failure on the 15th March, 1901.

Delirium with Delusions.

Case No 1, 1905.—T. H. Male, aged 13. Schoolboy. Admitted 22nd January, 1905. Discharged 26th February, 1905. Sudden onset with fever, vomiting, and headache. Next day noticed painful and tender swelling in right groin. Serum, 60 c.c.; later, 80 c.c. Right upper femoral bubo, which suppurated. Urine showed albumen for five days. Main symptoms: *Violent delirium with delusions for 3½ days.*

Corneal Ulcer.

* Case No. 18, 1901.—Male, age 2½ years. Admitted 12th May, 1901, on the 6th day of disease. Temperature on admission 102, pulse 150. Onset sudden, with convulsions. Glands affected: left cervical, right inguinal, right and left axillary. He had 1060 c.c. curative serum subcutaneously. Death on the 28th day. Prominent features were delirium, meningitis, *corneal ulcer.*

Keratitis.

* Case No. 41, 1902.—Aged 39. Living with her sisters at an hotel in Edward street. On the 3rd of February, 1902, one of the sisters (Case No. 38) died after what was thought a comparatively mild illness of four days' duration. *Post-mortem* examination revealed that she had died of bubonic plague.

Condition on Admission.—Temperature 103, pulse 120, soft; and respirations 36, shallow. Mind clear, cheek flushed, mosquito bites on face, arms, abdomen, and legs; conjunctivitis and keratitis of right eye.

As the condition of the right eye was giving trouble, Dr. Gibson was called in on the 14th. His notes, kindly placed at my disposal, are as follows:—

"Seen at Suspect Hospital on 14th and 21st February, and subsequently. Right eye suffering from infective conjunctivitis and keratitis. The keratitis took the form of general haziness of the cornea, superficial ulceration in its upper outer quadrant, with purulent infiltration of this quadrant chiefly at its periphery, and hypopion together with adhesive iritis. Duboisia failed to detach the iris from all its adhesions to the lens—a strong one being left superiorly and two weaker ones inferiorly. Bisulphate of quinine solution quickly caused a disappearance of the hypopion, and together with a tabloid of fluorescine daily, it led to clearing up of the purulently infiltrated cornea, and healing of its ulcerated surface. When discharged from the hospital, on 12th March, the cornea was clear, but a little pericorneal injection remained. Very slight pericorneal injection persisted for several weeks. Although intraocular tension did not appear to be increased, unless during the acute stage, some myopia resulted. When last seen on 4th June, vision was best with—2D, vision $\frac{6}{9}$ partly. The eye was probably emmetropic before the attack, though it is an assumption to say this and that the myopia was acquired, since no record of vision prior to the attack is available. The left eye is practically emmetropic, and sees $\frac{6}{6}$ partly."

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* "Report on Plague, Brisbane, 1901-2," by Dr. A. C. F. Halford.

Suppurating Corneal Ulcer.

Case No. 2, 1907.—Male, aged 39; sailor, out of employment. Admitted 14th January, 1907. Temperature 104, pulse 108, respirations 36. Bubo of right inguinal region. Conjunctivitis and keratitis of right eye, which subsequently developed into a large suppurating corneal ulcer. Had 120 c.c. serum. Discharged 16th April, after a prolonged convalescence.

Circulatory System.—The pulse rate varied in the initial stage from 75 to 150 per minute, but from the first it was peculiarly soft, full, and *dicrotic.* Marked dicrotism of the pulse associated with femoral adenitis during an epidemic of plague was regarded as almost pathognomic of the disease. An examination of the blood showed a considerable increase in the number of leucocytes, but the presence of plague bacilli was not often demonstrated except in pronounced septicæmic cases, or in bubonic cases as a sign of impending death. Dr. C. C. Baxter-Tyrie, late Health Officer to the Department, in an official report states:—

"With a view of ascertaining the value of the recent discovery of Bell, of Hongkong, that *B. pestis* is present in the blood of every case of plague, and not, as hitherto regarded, only in septicæmic cases or as an immediate antecedent of death, I have examined smears from the peripheral blood of the last eleven patients admitted to the hospital; I have found plague organisms present in the blood of eight. Three of these cases subsequently died, two from secondary septicæmic plague and one from secondary pulmonary infection, therefore the results obtained from them are not admissible.

"The presence of bacilli in the peripheral blood in the remaining cases, however, lends striking support to the view that there is in every case of plague a dissemination of bacilli throughout the general circulation. Of the three cases in which I failed to detect the bacillus, two were extremely mild, and the third showed a purely cutaneous lesion, which was treated in time to prevent even glandular infection. This accords with my previously expressed views that the severity of the disease, given the same degree of virulence, is directly proportionate to the number of organisms present. How soon the organisms appear in the blood I am unable to say until further investigation has been made. I have been unable to detect their presence after the defervescence of the initial pyrexia.

"This demonstration of the presence of bacilli as a common occurrence in the general circulation of individuals suffering from plague introduces a further tangible basis for doubting whether buboes are always indicative of local inoculation."*

Cases with large number of Bac. pestis in peripheral blood without severe constitutional symptoms.

Case No. 15, 1905.—Male, aged 25; saw-trimmer. Admitted 28th February, 1905; died 2nd March, 1905. Sudden onset on morning of 26th, with vomiting and fever; no diarrhœa or headache. On admission had 120 c.c. serum, and 120 c.c. next day. Albuminuria. Had abdominal pain on first day, with free perspiration. Showed signs of collapse about 4 a.m. on 2nd; died at 7.40 a.m. Main feature: Immense numbers of *B. pestis* in blood, without alarming symptoms.

Case No. 16, 1905.—G. Male (Chinese), aged 32; fruiterer. Admitted 6th March, 1905; died 7th March, 1905. Sudden onset on 4th, with severe headache, vomiting, and pains, and swelling in right groin. Had serum 120 c.c. on 6th and 120 c.c. on 7th. Had severe lumbar pains and albuminuria. Pulse became very weak and irregular before death. Main features: Immense numbers of *B. pestis* in blood on 6th, without any alarming symptoms.

The weakness of the circulation and the sudden failure of the heart's action have been explained by many observers as due to a myocarditis, or to a vasomotor paralysis by the plague toxin. As the heart returns to its normal condition more or less rapidly when sound health is regained, it is more probable that the weakness of the circulation is due to a toxæmia affecting the vagal centres and the intrinsic cardiac ganglia.

The Skin.—Ecchymotic patches and petechiæ were observed in several cases, but carbuncles, boils, and pustular eruptions were but rarely encountered. Epistaxis, hæmoptysis, and melæna were occasionally met with in septicæmic cases. The primary plague sore and the phlyctenule were observed in the following instances:—

Plague Lesions.

Case No. 17, 1904.—Produce hand. Initial temperature 104·2 degrees Fahr., pulse 90, weak and dicrotic. Serum, 120 c.c. subcutaneously. Temperature normal 8th day. Lower left femoral gland affected; sloughed *en masse.* Detained in hospital 46 days. The longest case. There was a *sore* on the inner side of the left calf, which yielded plague bacilli on examination. There was marked lymphangitis between the sore and the saphenous gland. The sore (circular, ¾-inch in diameter) consisted of a central black eschar, round which was a ring of purulent vesicles. Surrounding this again was an area of livid, brawny induration. From the same store as in Case 16.

Case No. 25, 1904.—Student. A remarkable case. Arrived in Brisbane a week previous to his attack. When seen by two medical practitioners he was found to have a *lesion* similar to malignant pustule on the nape of the neck, a central eschar within a zone of confluent pustules, and surrounded by an area of hard brawny indurations (*cf.* Case 17. "Plague lesions.") On bacteriological examination plague bacilli were found in large numbers. The lesion was energetically treated by incision and pure carbolic acid. Serum 240 c.c. was administered subcutaneously. The bacilli were evidently all eradicated in the local lesion as there was no secondary adenitis. Initial temperature 104 degs. Fahr., pulse 112, dicrotic; rapid constitutional recovery. Temperature normal 3rd day. There were no bacilli in the sore after treatment. The sore healed in a month.

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* "My method of making the smear was (all precautions were taken to eliminate possible contamination) to obtain very thin smears from the patient's blood by Cole's method. In a private communication from Dr. Bell, in which he furnishes further results, he informs me that he works with very thick smears which he decolorises. It is a matter of gratification that we have obtained similar results with diverse methods."

CASE No. 5, 1905.—Male, aged 59; packer in wholesale chemists. Admitted 5th February, 1905; discharged 25th March, 1905. Sudden onset with headache, vomiting, pain in abdomen and legs, and right groin. Anti-plague serum 160 c.c. Pulse weak and irregular. *B. pestis found in blister on leg.* Right femoral bubo, which suppurated. No albumen. Main symptoms: Stupor, delirium, cardiac weakness, and abdominal pain.

CASE No. 16, 1907.—Male, aged 40 years; labourer. Admitted 23rd January, 1907. Left axillary bubo. Had a *phlyctenule* on palmar surface of left forearm, in which *B. pestis* demonstrated. Died 30th January.

CASE No. 48, 1902.—Age 26 years, male. Admitted on 24th February, 1902, the 4th day of disease. Temperature on admission 103·2, pulse 114, respirations 28. Gland affected, left femoral. Duration of acute symptoms, 14 days. He had 160 c.c. curative serum subcutaneously and 100 c.c. intravenously. Prominent features were continued high temperature, restlessness, delirium, unilateral broncho-pneumonia with pleurisy, and *two deep abscesses.* Convalescence was slow and tedious, and he was discharged on the 19th April, 1902.

Petechiæ.

CASE No. 22, 1904.—Produce-store hand. Initial temperature 103·8 degrees Fahr., pulse 104, weak and dicrotic. Serum 480 c.c. subcutaneously. Left inferior external axillary glands affected, also left femoral Developed intense blood infection and secondary specific pneumonia, and died on the 10th day. The only case in 1904 where *subcutaneous hæmorrhages* were present; these appeared on the 6th day.

CASE No. 25, 1901.—Age 25 years, male. Admitted 28th May, 1901, on the 4th day of disease. Temperature on admission 102·8, pulse 104, respirations 28. Onset, sudden. Gland affected, left femoral. Had 100 c.c. curative serum subcutaneously. A trace of albumen was present. He collapsed suddenly, and died of heart failure three hours after admission. *Petechiæ* appeared on the upper part of his abdomen shortly before death.

Alimentary System.—Nausea and vomiting were constant symptoms at the onset of the illness. Occasionally abdominal pain was complained of. The bowels during the febrile period were constipated, diarrhœa being a symptom of the later stage.

The appearance of the tongue is somewhat characteristic of plague. In the first stage of the illness it is moist, with a creamy white fur along the dorsum, the tip and edges of the tongue being clean and of a bright pink colour. Later on it becomes dry, the fur a brownish or yellow colour, and the tip and edges deepening to a darker red or the colour of mahogany.

In the series of cases which occurred in the Rockhampton outbreak, several of the earlier cases had marked abdominal symptoms.

Genito-Urinary System.—During the initial pyrexia, the functional disturbance of the kidney is evidenced by the appearance of albumen in more or less quantity in the urine. Generally the amount is small and temporary, but, in fatal cases, albuminuria was often a marked symptom previous to the death of the patient. Hyaline and epithelial casts were frequently demonstrated in association with the albuminuria. Suppression was observed in one fatal case, but temporary retention was by no means unusual in the early period of the disease.

Pregnancy.—In pregnant women the most important complication is that of abortion. As a rule both mother and child die, but exceptions occur in which one or both may live.

CASE No. 17, 1901.—Age 25 years, female. Admitted 9th May, 1901, on the 5th day of disease. Temperature on admission 102·8, pulse 128. Onset, sudden. Gland affected, right inguinal. Duration of acute symptoms, 9 days. She had 500 c.c. curative serum subcutaneously. A trace of albumen was present. Prominent features were restlessness, delirium, persistent vomiting, and cardiac depression. The gland suppurated on the 9th day. She was discharged on the 3rd July, 1901. At the time of admission she was five months pregnant; at full term she gave birth to a healthy male child.

CASE No. 73, 1901.—Age 26. The patient was staying for a few days with her relatives in George street, city. While there her cousin (case 70) took ill, and, during what seemed a mild attack of illness, died suddenly, without having had any medical attendance. *Post-mortem* examination showed death to be due to plague.

The day before this event the patient returned to her home at Lawnton. She appeared quite well until Saturday, 29th March, 1902, when she had a shiver at 5·30 p.m. and again at 10·30 p.m. She was very restless and feverish all night, and had severe frontal headache. She remained in bed all next day complaining of severe headache and pain in the left groin, where she noticed a slight swelling, which was tender.

On the 31st the swelling in the groin was larger and more tender. She was very thirsty and feverish all day, and the headache was still severe. She vomited once during the afternoon, and at 10 p.m. she was confined of a male child, which was dead. Her case was reported by a doctor, who sent a specimen from the bubo to Brisbane, where it was found to contain plague organisms plentifully. A nurse was sent up from Brisbane to look after her on the 1st April. The nurse reported her condition as follows:—At 6 p.m. temperature was 104·4, pulse 138, and respirations 40. Tongue very dry and brown. There was a brownish vaginal discharge which was very offensive. There was an enlarged gland in the left inguinal region and one in the left femoral region. The skin over them was much reddened, and there was a good deal of inflammation around them. They were extremely tender. At 7 p.m. she had 100 c.c. of curative serum subcutaneously into the left thigh. She was delirious all night, and slept only at short intervals. Next morning her temperature was 103·6, pulse 140, and respirations 34. She seemed a little brighter and more sensible, but later became very restless and delirious. The vaginal discharge still continued to be very offensive. At 9 a.m. she had 80 c.c. of curative serum subcutaneously. The nurse then returned to Brisbane and reported the condition of the patient. On returning to Lawnton in the afternoon she was supplied with the necessary appliances for uterine irrigation, and also with a further supply of curative serum. She found the patient at 6 p.m. with a temperature of 104·8, pulse 128, and respirations 26. At 7 p.m. 100 c.c. of curative serum was given subcutaneously. At 8 p.m. the uterus was douched first with lotion of hyd. perchlor. 1:1000, followed by a copious, weak lysol douche. She was very restless and delirious until about 1 a.m., after which she slept fairly well. Next morning the temperature was 102·4, pulse 118, and respirations 30. Patient seemed a little better, and mind clearer, although she was delirious at times during the day. During this time she had been douched at regular intervals. On the evening of the 3rd she was transferred to the Suspect Hospital, where she arrived at about 12 midnight. Her temperature was found to be 102·8, pulse 104, and respirations 28. She slept well until morning; her temperature had then fallen to 99, pulse 110, and respirations 28. She was then curetted, and a quantity of placental *débris* and blood-clot was removed. From this out she progressed well and made an uninterrupted recovery, except that both glands suppurated and were opened and each gland sloughed away, leaving a healthy wound which soon closed. She was discharged on the 17th April, and by the end of the month appeared to be in perfect health.

Case No. 28, 1907.—Female, age 23, household duties. Admitted to the Lady Bowen Hospital, where she was confined of a full-term child on 5th April, 1907. Died on 8th April. The infant, a male, lived seven days. Died on 12th April. *Post-mortem* examination revealed presence of *B. pestis.*

Respiratory System.—The respirations in all types of the disease are increased in frequency, the pulse-respiration ratio being invariably disturbed, even in cases where no lung symptoms were apparently present. The case of primary plague pneumonia given below was remarkable for its high respiration rate.

Secondary pneumonia supervened in a certain proportion of septicæmic cases, and, although not regarded as so inevitably fatal as the primary pneumonia, was always looked upon as a serious complication.

Case No. 3, 1905.—Female, age 22 years, nurse. Attended two fatal primary pneumonic cases. She became ill on 3rd June, 1905, with severe headache and malaise; 80 c.c. of serum subcutaneously. On 4th June showed symptoms of pneumonia. Temperature 104. On the morning of the 5th June the pulse had increased to 148, and the respiration to 48. No glandular enlargements. On the afternoon of the 5th the temperature was 104, pulse 156, respiration 54. Sputum fairly abundant and slightly pink in colour. Bacterioscopic examination of sputum showed *B. pestis*, pneumococci, and streptococci. During the evening she rapidly became worse, the respiration being increased to 60, the pulse to 160, while the temperature rose to 105. She died early on the 6th June.

II.—TYPES OF PLAGUE.

1.—"PESTIS MINOR."

The term "*Pestis Minor*" is commonly understood to mean mild, minor, or benign plague, including the abortive and ambulatory forms. Many instances of mild forms of the ordinary bubonic type have come under our notice from time to time during an epidemic of the latter, but *epidemic pestis minor* has not so far been observed in Australia. It has, however, been observed to prevail in other parts of the world. A non-fatal malady characterised by glandular swellings, with more or less fever and malaise, preceded or ran concurrently with a well recognised epidemic of plague in the city of Astrakhan in 1877.

A non-fatal form of plague was described by Sydenham as observed in London in 1665-66, and by Fodin, under the name of "benign plague," as occurring in the Levant in the early part of the last century.

Dr. J. F. Payne (System of Medicine, 1907. Vol. II. B. Clifford Allbutt. Page 388) mentions the fact that the Eastern Asiatic strain of plague is distinguished by the frequent occurrence of epidemics of *pestis minor* or mild plague, as contrasted with the Indo-Chinese strain in which the epidemics of *pestis minor* are of less frequent occurrence. As the present pandemic of plague distributed widely over the world is, however, the Yunnan plague of Southern China, the points of difference between the two strains are not so distinct as they formerly appeared, and probably are of more historical than clinical interest.

Cantlie (System of Medicine, Allbutt, 1907) describes a disease, chiefly occurring in tropical and subtropical countries, under the name of "Climatic Bubo." It is characterised by the development of buboes of non-venereal origin in the inguinal region in the glands of either the oblique or vertical group, the appearance of the bubo being associated with a rise in temperature with marked weakness. Suppuration of the bubo is the rule in all cases, the swelling rapidly increasing to the size and shape of half an orange by the fourth or fifth day. It is not a fatal disease.

The name "*pestis minor*" was given to this complaint by Cantlie, without any idea of indicating mild, ambulatory, or abortive cases of plague thereby.

Pestis minor is regarded by this observer as synonymous with "climatic bubo," and is intended to indicate a specific ailment—anticipating, running synchronously with, and continuing after outbreaks of true plague—but still preserving its own character. Variation in the severity of the disease (the occurrence of symptoms not mentioned in descriptions of the standard type of plague, or characteristised by restriction in the number of symptoms rather than by increase of additional symptoms) has apparently produced the many varieties of benign plague recorded by earlier observers. If the expression *pestis minor* is commonly understood to mean not a mild case of true bubonic plague, but a specific form of glandular enlargements with fever, which occurs only when plague threatens or is present, then the term is a most misleading one. The fact that the *B. pestis* has occasionally been met with in buboes in so-called *pestis minor* cases, and the fact that such cases have sporadically occurred during an epidemic of true bubonic plague, lead to the conclusion that either these cases are really mild plague, and, therefore, appropriately, or rather, conveniently, designated "*pestis minor*," or minor plague; or that they are merely cases of glandular enlargements not of a bubonic plague type, and, therefore, erroneously called "*pestis minor*."

If *pestis minor* be not true plague, the term should be dropped altogether as applied to buboes of a non-specific type. This contention was prominently brought forward by me at the Medical Congress of Australasia, held at Hobart in February, 1902. A resolution to the effect that the term "*pestis minor*" should not be applied to non-plague cases met with the unanimous approval of the full Congress. In this, the mildest type of the bubonic form, the patient suffers from headache, malaria, slight pyrexia, pain and tenderness in the groins and armpits associated with slight glandular swellings. There are also usually vomiting, nausea, slight congestion of the eyes, and sometimes slightly impaired speech.

In the more acute form buboes develop, which terminate in resolution or suppuration. Plague bacilli were frequently found in the bubo-material, though it occasionally happened that the patient was well and about his ordinary avocation before the inoculated guinea-pig experimented upon had died with the symptoms of plague. This fact will be adverted to in connection with the subject of clinical bacteriology.

Reference has been made to the fact that *epidemic pestis minor* did not come under our observation. The outbreak at Mossman, Port Douglas, in the North of Queensland, early in the year 1907, furnished a group of symptoms, which were interpreted by the local health officer at Port Douglas to be consistent with *pestis minor*. The epidemic extended over four or five months and in all some sixty cases of so-called *pestis minor* were reported. There were only two deaths. In only ten of the cases, however, did the bacteriological evidence prove confirmatory of plague. All the patients were cane-cutters, living in encampments within a few miles of the sugar plantations upon which they were working. A curious group of symptoms was observed by Dr. O'Brien, late Health Officer at Cairns, who investigated many of the cases on behalf of the Department. The illness lasted only a few days, and was characterised by more or less general enlargement of the lymphatic glands, especially in the groin and armpits, without the formation of any definite buboes. The smallness of the glandular swellings and their non-suppuration, the more or less active delirium of the patients, *subsultus tendinum*, a remarkably slow pulse, and the low death rate were conspicuously apparent in a considerable number of the cases observed by Dr. O'Brien, an officer experienced in plague work. He was of the opinion that the majority of cases observed by him were of the *pestis minor* type of plague. Sporadic cases continued to be reported up to the end of May, and early in that month I despatched the health officer (Dr. Chesson), of the Department, for the purpose of further investigation. His report, made on the 13th June, 1907, states that—"There were six cases under observation in the Port Douglas Hospital at the time of my arrival. Most of the superficial glands were 'shotty,' and, perhaps, slightly larger than normal; but there were no buboes, tenderness, nor periadenitis. I took specimens of lymph from the most prominent glands I could find, from which films were prepared and culture tubes and guinea-pigs inoculated; but all three tests—microscopical, cultural, and experimental—were plague negative. An examination of the blood of many patients also gave negative results. . . . From information volunteered by people who have resided for many years in Port Douglas and Mossman districts, it appears that similar outbreaks of sickness amongst workers in the canefields and bush are by no means uncommon, especially in seasons marked by excessive rainfall, as was the case in the early part of this year. I, with Mr. Beardmore, of the Bacteriological Institute, examined all rats caught during my stay in the district, but no evidence of plague was obtainable, nor was there a history of great mortality among the rats. . . . . I am of opinion that this epidemic was of the nature of one of those unclassified fevers so often met with in tropical climates, and the history and temperature charts in many of the cases resembled closely what has been described as 'double continued fever.'" Since the end of May no further cases have been reported from the district. Still, the fact remains that in some of the cases (10) bacilli morphologically resembling *B. pestis* were observed.

1. Examples of *mild* cases of bubonic plague are given below:—

* Case No. 92, 1902.—A woman, aged 40. Sickened on the 24th, the illness being ushered in with repeated shivering and also a good deal of vomiting. She also complained of giddiness and headache.

On the 25th she complained of pain in the groin, and noticed a small swelling there. The pain was very severe for about twenty-four hours, but after that the swelling was only tender when touched.

On the 26th, when seen she was complaining of headache, pain in the abdomen, and about the body, especially about the lumbar region, and thirst She was of somewhat nervous temperament, but quite rational; speech rather slow and hesitating. Face and neck exceedingly flushed, eyes somewhat injected, tongue dirty white on dorsum, red at tip and edges, faucial congestion and dryness. Pulse, full and bounding, about 100; temperature, 103·4; respiration, 28. She had a moderately enlarged gland in the right femoral region, with a little periadenitis. Skin over it reddened. Sero-lymph from gland bacteriologically negative.

On the 27th the patient was about the same, skin dry, and she was very thirsty. Temperature, 103·6 in a.m. and 103·8 in the evening. Pulse, somewhat dicrotic, about 106. The gland was larger, and periadenitis had increased. Specimen taken bacteriologically positive.

On the 28th, at 3 p.m., quite bright and rational, speech somewhat slow and thick. Temperature, 102·6. Face and neck brightly flushed, eyes rather injected, tongue red and dryish, pulse small and soft, being 94, and respiration 22. Chest clear, abdomen nothing abnormal, has somewhat enlarged tender gland in right femoral region, with a good deal of periadenitis.

At 6 p.m. temperature 104, pulse 98, and respiration 28. At 7.30 p.m. 120 c.c. curative serum subcutaneously right abdomen. After the serum injection she slept well all night.

Temperature in the morning was 102 and pulse 100. Her face was much less flushed and her tongue moister. The temperature rose during the afternoon to 102·8, pulse 108, and respiration 24 at 4 p.m., but her general condition remained unaltered. She slept well that night, and next morning her temperature was 100. After this she progressed uneventfully, except that her bubo suppurated and was opened on the 2nd May. It contained a small amount of thin reddish pus, which contained degenerated *B. pestis*. It rapidly healed, and she was discharged on the 28th May.

Case No. 35, 1907.—Male, aged 45. Licensee of hotel where two previous cases occurred. Onset 26th May. Temperature, normal; pulse, 80. Left femoral bubo. *B. pestis* found in "specimens" after death of the inoculated guinea-pig. Patient was convalescent when visited by Health Officer on 31st May.

II.—SEPTICÆMIC TYPE OF PLAGUE.

Case No. 12, 1901.—Age 38 years, male. Admitted to General Hospital 12th April, 1901, on the 4th day of disease. Temperature on admission, 104·6; pulse, 130; respirations, 36. Patient stated that he was quite well until the 9th April. On that day he complained of severe headache, and in the evening vomiting. The next day the headache was more severe, and he had pains in his abdomen. On the 11th he noticed a painful swelling in his left groin. Patient arrived at hospital spitting up blood, and walked up to the ward. There was no delirium, and he died of heart failure a few hours after admission. He had no serum administered.

Case No. 10, 1904.—Male, 16 years. Produce hand. Initial temperature, 99·2 degrees Fahr. Serum 120 c.c. injected subcutaneously. No glands. Died on the second day. General blood infection. Bacilli were mixed in type, but mainly coccal.

Case No. 15, 1904.—Male, aged 22 years. Oyster-saloon cook. Infected rats from the shop this year (1904). Four days ill before seen. Initial temperature, 105·4 degrees Fahr. No glands. Pulse, 152 (dicrotic). Serum 120 c.c. subcutaneously. Died shortly after being seen by Health Officer. General blood infection.

Case No. 18, 1904.—Male, 30 years. Sanitary carter. Ill three days. Died before arrival of Health Officer. Autopsy: General blood infection and secondary pneumonia. Retroperitoneal glands affected.

Case No. 22, 1904.—Male, 25 years. Produce store hand. Initial temperature, 103·8 degrees Fahr.; pulse, 104 (weak and dicrotic). Serum 480 c.c. subcutaneously. Left inferior external axillary glands affected, also left femoral. Developed intense blood infection and secondary specific pneumonia, and died on 10th day.

Case No. 15, 1905.—Female, aged 20. Domestic. Patient admitted to Brisbane General Hospital on 2nd February, 1905. Her temperature on admission was high, and it was noted in the hospital case records that the pulse was dicrotic and the spleen enlarged. There was no external bubo. Before death the temperature reached 107 degrees Fahr., the pulse was uncountable, and the respirations 40. P.M. examination showed a general enlargement of the lymphatic glands, and the mesenteric glands were enlarged and hæmorrhagic. Cultures from these glands showed typical *Bac. pestis.*

Case No. 16, 1905.—Male, aged 14. Produce store hand. Onset sudden, with malaise, headache, and vomiting. No glands. Was delirious next day. *Bac. pestis* in considerable numbers found in the blood stream. Had 200 c.c. of Yersin's serum injected altogether. Died two days after admission to the hospital.

III.—PRIMARY PLAGUE PNEUMONIA.

Case No. 1m.—R. O'C., male, aged 10. Schoolboy. One of a family of seven. Four members of the family died of septicæmic plague, with secondary pneumonic symptoms, previous to his death on 2nd June, 1905. Sudden onset on 30th May, 1905, with rigor, headache, and vomiting. Temperature, 104; pulse, 126; respirations, 42.

On 31st May felt worse; precardial pain, cough, moist rales at apex of left lung. Delirium; involuntary micturition. 1st June signs of pneumonia heard below left clavicle; sputum scanty. No glandular enlargements. Became steadily worse during the night; sputum now loose and watery. Died at 2.20 p.m., 2nd June, 1905.

Post mortem.—Upper two-thirds of left lung dark and solid, red hepatisation being well marked. Sections sank in water. Several hæmorrhagic patches over middle lobe of left lung. Sero-sanguineous fluid exuded from mouth and nostril and filled bronchial tubes. No glandular enlargements. Liver large, tough, and intensely hyperæmic. Bacteriological examination of sections of lungs showed numerous *Bac. pestis.* A guinea-pig inoculated with material from lungs died in four days, and numerous typical plague bacilli were recovered from the spleen. The sputum of the patient which had been collected before death contained a large number of *Bac. pestis.*

Case No. 2r.—R. W., female, aged 28. Nurse attended above case. Was taken ill on 8th June, 1905, with symptoms of lobular pneumonia, cough, and copious watery sputum. Temperature, 105; pulse, 124; respirations, 23. The sputum contained enormous numbers of *Bac. pestis.* She died on 12th June, at midnight.

Case No. 28, 1901.—Aged 30, living at South Brisbane, was admitted to the Brisbane General Hospital on the 4th June, 1901. The patient was quite well until the 28th May, 1901, when he complained of headache and feverishness. Next day he complained also of chilliness, headache, feverishness, and weakness. He continued in the same condition until the 1st June, when he got up and went for a walk, feeling better. He had on some previous occasions suffered from fever contracted in New Guinea, and put his present illness down to the same cause. He got worse again that night, and remained in bed all next day. On the 3rd he complained of pains all over his body. On the 4th the pains were more severe, and the breathing became rapid and his voice husky. He was seen by a medical man, who noticed a rash like that of measles about the body, but more especially marked on the arms. The eyes were injected and the patient had a slight cough. A provisional diagnosis of measles was made, and the patient removed to the General Hospital. On admission his temperature was 103, pulse 140, and respirations 60. No bronchial breathing could be detected, but there were moist sounds from base to apex on both sides. He only lived a few hours in the hospital. He had no serum, and at the *post-mortem* examination the whole of both lungs was found extensively consolidated. The bronchial glands were enlarged and blackened with hæmorrhagic inflammation. No enlarged glands were found anywhere else, nor was there any other macroscopic changes noted in the other organs. Smear preparations from the affected lungs showed presence of innumerable plague bacilli apparently in pure culture. An infected rat was found at the place of his employment

Case No. 35, 1901.—Age 33 years, male. Admitted to General Hospital in a state of collapse 2nd October, 1901, on the 5th day of disease. Pulse, 84; respirations, 48. His temperature was under 95. He was spitting up a great deal of thin blood-stained sputum in which plague bacilli were very numerous. There were moist sounds all over both lungs. He had no serum administered, and died five hours after admission.

IV.—INTERESTING FEATURES IN CONNECTION WITH DURATION OF SYMPTOMS.

Case No. 30, 1901.—Age 19 years, male. Admitted 28th June, 1901, on the 4th day of the disease. Temperature on admission, 103·8; pulse, 118. Glands affected, left femoral and right inguinal. Onset, sudden. Had 720 c.c. of curative serum subcutaneously. The left femoral gland suppurated on the 7th day. On the 3rd July both submaxillary glands, a gland in each axilla, and one in the right inguinal region, became affected. On the 5th July he developed three occipital glands. On the 6th July the right parotid lymphatic gland, on the 7th July the right and left concatenate glands and the right and left supratrochlear glands became affected. Prominent features were: Continued high temperature and delirium, marked and continuous cardiac depression. Vivid and persistent serum rashes developed, followed by universal exfoliation. He lay in a critical typhoid-like state for almost two months, and his subsequent convalescence was very tedious. He was discharged in a fairly good state of health after about three months' stay in the hospital.

Case No. 46, 1902.—Age 19 years, male. Admitted on 20th February, 1902, on the second day of disease. Temperature on admission 101, pulse 120, respirations 24. Onset sudden. The glands affected were left inguinal and femoral. Duration of acute symptoms, six days. He had 240 c.c. curative serum subcutaneously and 20 c.c. of fresh ox serum also subcutaneously, and 210 c.c. of curative serum intravenously. Prominent features were: Double broncho-pneumonia, restlessness, delirium, and marked cardiac depression. The inguinal gland suppurated on the 8th day, and the whole chain of inguinal glands sloughed away, leaving a large sinus, which did not heal for about two months; $\frac{3}{4}$-albumen was present.

Case No. 3, 1903, lived for eighty-one days after admission to the Plague Hospital, during the whole of which time intractable diarrhœa, with involuntary evacuations, persisted, resulting eventually in bed sores which, with their inevitable constant pain, sleeplessness, and sapræmia, materially hastened the end.

The temperature throughout was extremely high, scarcely ever being below 102 degrees until within eight days of death, and ranging daily up to 103-4-5 degrees, resisting alike serum, baths, quinine, and anti-pyretic drugs. This sapræmia, and, later, septicæmia, were accounted for by finding eventually that the chain of glands extending from the upper femoral deep regions to the brim of the pelvis behind the peritoneum had broken down into what was practically a tunnel full of pus and sloughing broken-down glands, from which a pure culture (morphologically) of *Bacilli pestis* was recovered at the autopsy. From the urine at the same time was obtained a pure culture of the same organisms.

III.—CLINICAL NOTES.

The following abstracts of the more interesting cases treated at the Plague Hospital, Brisbane, exhibit the salient features of each case. The notes have been supplied by the various Health Officers in charge of the patients from time to time:—

1901.

[Excerpts from a "Report on Plague, Brisbane, 1901-2," by Dr. A. C. F. Halford, late Medical Officer to the Metropolitan Joint Board.]

Case No. 1.—Age 21 years, male. Admitted 28th February, 1901, on the 4th day of disease. Temperature on admission 104·8, pulse 110, respirations 26. Onset, gradual. Gland affected, right femoral. Duration of acute symptoms, 4 days. Had 80 c.c. curative serum subcutaneously. Prominent feature was rapid reaction to serum. He was discharged on the 29th March, 1901.

Case No. 6.—Age 29 years, male. Admitted 23rd March, 1901, on the 2nd day of disease. Temperature on admission 103·8, pulse 96, respirations 24. Onset, gradual. Gland affected, right femoral. $\frac{1}{4}$ albumen was present. Gland suppurated on the 7th day. Duration of acute symptoms, 6 days. Discharged on the 22nd April, 1901. 180 c.c. of curative serum administered subcutaneously.

Case No. 7.—Age 10 years, female. Admitted 28th March, 1901, on the 2nd day of disease. Temperature on admission 104·4, pulse 162, respirations 30. Onset, gradual. Gland affected, right femoral. She had 364 c.c. curative serum subcutaneously, and 40 c.c. curative serum intravenously. Prominent features were delirium, restlessness, continued high temperature, and death from heart failure on the 6th day. A trace of albumen was present.

Case No. 8.—Age 11 years, male. Admitted 30th March, 1901, on the 3rd day of disease. Temperature on admission 104·4, pulse 126, respirations 28. Onset, sudden. Gland affected, left femoral. Duration of acute symptoms, 4 days. He had 170 c.c. curative serum subcutaneously. Prominent features were restlessness, delirium, and marked reaction to serum. The gland suppurated on the 6th day. He was discharged on the 29th April, 1901.

Case No. 19.—Age 26 years, male. Admitted 9th May, 1901, on the 5th day of disease. Temperature on admission 103·6, pulse 108, respirations 20. Onset, sudden. Gland affected, left inguinal. Duration of acute symptoms, 5 days. He had 200 c.c. curative serum subcutaneously. Prominent features were delirium, marked cardiac depression, and pronounced reaction to serum. Discharged 10th June, 1901.

Case No. 24.—Age 17 years, male. Admitted 20th May, 1901, on the 2nd day of disease. Temperature on admission 105·8, pulse 124. Onset, sudden. Gland affected, left femoral. He had 820 c.c. curative serum subcutaneously. On the 6th day he developed fresh glands in the right inguinal region, and right and left submaxillary regions, and died of heart failure on the 8th day. The prominent features were coma, restlessness, and delirium.

Case No. 29.—Age 24 years, male. Admitted to General Hospital, 25th June, 1901, on the 5th day of disease. Temperature on admission 104, respirations 24. Patient stated that he first complained of severe headache on the 21st, with pain in the abdomen and lumbar region, and continued in this condition until admission. On the 26th he began spitting up blood-stained sputum, and on examination the bases of both lungs were found to be full of moist sounds. On the evening of the 26th the moist sounds could be found nearly up to the apices on both sides. During the night he became very delirious and got out of bed. On the morning of the 27th the cervical glands on both sides were found to be much enlarged. Breathing became very difficult, and tracheotomy was performed to relieve this condition, which it did for a short time, but he died of heart failure about the middle of the day. He had no serum, as the diagnosis was not made until shortly before death.

Case No. 34.—Age 47 years, male. Admitted 16th August, 1901, at 3·45 p.m. Temperature on admission 101·4, pulse 134, respirations 40. He was in an unconscious and collapsed condition, and unable to give history. He had a bubo on the left femoral region, and a small gland in the right and left axilla. He never regained consciousness, and died at 9 p.m. on the 16th August. He had 100 c.c. curative serum subcutaneously.

1902.

[Excerpts from Dr. Halford's Report.]

Case No. 40.—Age 33 years, male. Admitted to the Brisbane General Hospital on the 5th February, 1902. Temperature on admission 104, pulse 100, respirations 32. Patient stated he was quite well until the 2nd February, when he complained of pains in his head, abdomen, and legs, and severe vomiting. Since then the pains had gradually become more severe. Patient was treated as typhoid fever, and died on the 9th. The diagnosis of plague was made P.M. He had no serum. Gland affected, right inguinal.

Case No. 42.—Age 11½ years, male. Admitted 17th February, on the 2nd day of disease. Temperature on admission 103·4, pulse 120, respirations 30. The following glands were affected: two in the left inguinal region, one in the right, and one in the left submaxillary, and one in the left axillary. Duration of acute symptoms, 4 days. Had 100 c.c. of curative serum intravenously, and 100 c.c. curative serum subcutaneously, and 40 c.c. of fresh ox serum. The prominent features in his case were restlessness and delirium. He was discharged on the 15th March, 1902.

Case No. 45.—Age 11 years, male. Admitted 15th February, 1902, on the 7th day of disease. Temperature on admission 104·6, pulse 128, respirations 30. Onset, sudden. Glands affected in the right and left inguinal and femoral regions, right supratrochlear, and right submaxillary regions. Duration of acute symptoms, 9 days. He had 160 c.c. curative serum subcutaneously. The prominent features were continued high temperature, restlessness, and delirium. The submaxillary gland suppurated on the 25th day. No albumen was present. He was discharged on the 31st March, 1902.

Case No. 52.—Age 14 years, male. Admitted 5th March, 1902, on the 3rd day of disease. Temperature on admission 103·4, pulse 120, respirations 32. Onset, sudden. Glands affected, left inguinal, right inguinal, and left axillary. Duration of acute symptoms, 6 days. He had 200 c.c. curative serum subcutaneously and 240 c.c. intravenously. Prominent features were restlessness and delirium. ⅛ albumen was present. The left inguinal gland suppurated on the 10th day, and was accompanied by a great deal of sloughing, which left a large sinus. This healed slowly, and was accompanied by œdema of the left leg. He was discharged on the 26th April, 1902.

Case No. 53.—Age 22 years, male. Admitted 6th March, 1902, on the 3rd day of disease. Temperature on admission 103·4, pulse 120, respirations 20. Onset, sudden. Gland affected, left inguinal. Duration of acute symptoms, 3 days. Serum injected, 130 c.c. intravenously. Gland suppurated on the 7th day. The prominent feature was rapid reaction to serum. A trace of albumen was present. He was discharged on the 5th April, 1902.

Case No. 60.—Age 17 years, male. Admitted 10th March, 1902, on the 2nd day of disease. Temperature on admission 105·4, pulse 120, respirations 36. Onset, sudden. Glands affected, left femoral and left inguinal, and right and left axillary. Duration of acute symptoms, 3 days. He had 60 c.c. curative serum subcutaneously, 260 intravenously. Prominent features were coma, restlessness, and delirium, and marked reaction to serum. A trace of albumen was present. Left femoral gland suppurated on the 12th day. He was discharged on the 16th April, 1902.

Case No. 62.—Age 17 years, male. Admitted 11th March, 1902, on the second day of disease. Temperature on admission 103·4, pulse 80, respirations 24. Onset, sudden. Gland affected, right femoral. Duration of acute symptoms, 3 days. He had 200 c.c. curative serum subcutaneously and 140 c.c. intravenously. Prominent features were delirium and rapid reaction to serum. The gland suppurated on the 5th day. No albumen was present. He was discharged on the 12th April, 1902.

Case No. 65.—Age 27 years, male. Admitted 19th March, 1902, on the 2nd day of disease. Temperature on admission 103·4, pulse 138, respirations 34. Onset, sudden. The glands affected were right and left inguinal and femoral, right and left submaxillary. Prominent features were double broncho-pneumonia, delirium, restlessness, and continued high temperature. Serum injected, 1,780 c.c. intravenously. Femoral and inguinal glands suppurated on the 12th day; left submaxillary on the 13th day. ¼ albumen was present. He died on the 4th April, 1902, at 3·50 p.m.

Post Mortem Notes.—

P.M. 5th April, 1902. Very powerfully built, well-nourished man. Rigor mortis present. Posterior hypostasis well marked, none anteriorly. He has a mass of suppurating glands in left inguino-femoral region and right inguinal region, and left cervical region. Right cervical region glands enlarged, pinkish, but no suppuration.

Thorax.—Pericardium contained a little clear fluid. Heart large, pale, flabby, and soft; walls of right ventricle somewhat thin. Pleural sacs contained a little blood-stained fluid. Numerous recent adhesions on both lungs at base, anteriorly and posteriorly. Lungs pale anteriorly, somewhat engorged posteriorly, both bases very œdematous. Numerous round abscesses about ¾-inch in diameter, scattered through both lungs in substance and beneath pleuræ, contained yellowish pus. Bronchial glands not enlarged.

Abdomen.—Fat on walls about 2 inches deep. Peritoneum normal. Stomach and intestines pale, and somewhat distended with gas. Spleen much enlarged, purplish black in colour, and almost confluent. In the substance were numerous pin point abscesses with yellow pus. Liver enlarged slightly and congested, normal consistency, no abscesses. Kidneys enlarged, congested, and somewhat tough, with capsule adherent. Mesenteric glands not enlarged.

Bacteriological Examination.—

The spleen, liver, lungs, and kidneys proved sterile. *B. pestis* were demonstrated in smear preparations of the pus from lungs and spleen, but they were apparently dead.

Case No. 67.—Age 18 years, male. Admitted 21st March, 1902, on the 6th day of disease. Temperature on admission 102, pulse 104, respirations 30. Onset, sudden. Glands affected, right and left inguinal, right and left femoral, right and left submaxillary, and right and left axillary. Duration of acute symptoms, 33 days. Prominent features were restlessness, delirium, double broncho-pneumonia, and superficial abscess. The right submaxillary gland suppurated on the 18th day. A trace of albumen was present. He died on the 17th April, 1902, at 8 p.m. Had serum 480 c.c. subcutaneously, and 870 c.c. intravenously.

Post Mortem Notes.—

P.M. 18th April, 1902, at 7 a.m. Well-developed but poorly nourished. Rigor mortis present, well marked. Body pale; some posterior hypostasis, none anteriorly. There was a mass of suppurating glands on each side of the neck. The femoral glands on both sides were enlarged and hard.

Thorax.—Pericardium contained about 2 ounces of clear fluid. Heart pale, left ventricle contracted, walls firm and thick; right ventricle dilated, walls very thin. The large veins were filled with dark fluid blood. The pleural cavities contained a small quantity of clear fluid, no pleurisy present. Lungs pale anteriorly, posteriorly some venous congestion. Scattered through both lungs, under the pleuræ and in the substance, were numerous round abscesses, from 1 inch to ½-inch in diameter, with fairly tough walls. They contained thick yellow pus. The bronchial glands were enlarged and pinkish on section.

Abdomen.—The peritoneal cavity contained about 2 ounces of clear fluid, the peritoneum appeared normal. The stomach and intestines were pale and somewhat distended with gas. The liver was somewhat pale and toughened. The spleen was decidedly enlarged, of reddish purple colour, and quite pulpy. It contained two round abscesses, one about $\frac{1}{2}$-inch in diameter, the other $\frac{1}{4}$-inch. The pus in them was thick and yellow. Kidneys decidedly large, venously congested. The capsule was somewhat adherent and substance toughened. Mesenteric glands were not enlarged.

Bacteriological Examinations.—

The spleen, liver, lungs, and kidneys proved sterile. *B. pestis* were demonstrated in smear preparations of the pus from the abscesses in the lungs and spleen, but they were apparently dead.

Case No. 70.—Age 16 years, female. Died on 30th March, 1902. Duration of illness, about 7 days. Had no treatment. Diagnosed P.M. Had femoral gland.

Case No. 71.—Age 21 years, female. Died 30th March, 1902. Duration of illness, 30 hours. Had no treatment. Diagnosed P.M. Had gland in left femoral region.

Case No. 76.—Age 25 years, male. Admitted 11th April, 1902, on the 7th day of disease. Temperature on admission 101·8, pulse 110, respirations 42. Onset, sudden. Glands affected in the right and left cervical region, right and left submaxillary region, and right axillary region. He had double broncho-pneumonia. He had 120 c.c. curative serum subcutaneously, and died of heart failure a few hours after admission.

Case No. 82.—Age 45 years, male. Admitted 19th April, 1902, on the 8th day of disease. Temperature on admission 101·4, pulse 92, respirations 24. Onset, sudden. Glands affected, left inguinal, and right and left submaxillary. Duration of acute symptoms, 9 days. He had 240 c.c. curative serum subcutaneously, and 80 c.c. intravenously. 1-7th albumen was present. The left inguinal gland suppurated on the 13th day. There was a good deal of sloughing, and a deep sinus remained, which healed slowly. He was discharged on the 24th May, 1902.

Case No. 86.—Age 60 years, male. Died on 22nd April, 1902. Primary pneumonic, no glands. Duration of illness, 7 days. Seen in his own home, and had 160 c.c. curative serum subcutaneously a few hours before death.

Case No. 88.—Age 16 years, male. Admitted 24th April, 1902, on the 1st day of disease. Temperature 102, pulse 124, respirations 26. Onset, sudden. Duration of acute symptoms, 1 day. He had 60 c.c. curative serum subcutaneously. A trace of albumen was present. Prominent feature was rapid reaction to serum. He was discharged on the 17th May, 1902.

Case No. 90.—Age 50 years, female. Died 25th April, 1902. Bubonic. Duration of illness, some days, not exactly known. Had no treatment; diagnosed P.M.

Case No. 91.—Age 15 years, male. Admitted 25th April, 1902, on the 4th day of disease. Temperature on admission 104·6, pulse 102, respirations 24. Onset, sudden. Gland affected, right femoral. Duration of acute symptoms, 5 days. He had 240 c.c. curative serum subcutaneously. Prominent features were restlessness and delirium, and rapid reaction to serum. The gland suppurated on the 9th day. He was discharged on the 2nd June, 1902.

Case No. 94.—Age 11 months, male. Admitted in a dying condition, 27th April, 1902. Temperature on admission 103, pulse 88. Duration of illness, unknown. Had 100 c.c. curative serum subcutaneously. Died 2 hours after admission, at 2 a.m.

Post Mortem Notes.—

P.M. 27th April, 1902, at 11 a.m. Very well-developed, well-nourished body. Body pale; some posterior hypostasis, none anteriorly. In the right inguinal region there was an enlarged gland surrounded by a good deal of hæmorrhagic serous effusion. There were several small glands in femoral region on both sides. On section the glands were of pinkish colour, and showed numerous points of hæmorrhage.

Thorax.—Heart well developed, muscle rather soft. Left side firm and contracted, right dilated. Thymus was well developed. Bronchial glands were not enlarged.

Abdomen.—There was a layer of fat one inch thick on anterior abdominal wall. The peritoneum was normal. The stomach and intestines were pale. The spleen was somewhat enlarged, of reddish-purple colour, and fairly firm consistency. The kidneys showed some subcapsular hæmorrhage and congestion of the cortices, otherwise normal. The liver was deeply venously congested, of normal consistency. The mesenteric glands were enlarged and pinkish on section. The retroperitoneal glands were not enlarged.

Bacteriological Examination.—

Affected glands and spleen were examined, and showed the presence of typical *B. pestis.*

Case No. 100.—Age 27 years, male. Admitted 1st May, 1902, on the third day of disease. Temperature on admission 103·4, pulse 120, respirations 38. Onset, sudden. Gland affected, left cervical. Had 240 c.c. of curative serum subcutaneously and 640 intravenously. Prominent features were double broncho-pneumonia, pleurisy, restlessness, and delirium. Death occurred on the 7th May, 1902, at 6 a.m.

Post Mortem Notes.—

P.M. 7th May, 1902, at 11 a.m. Well-developed, well-nourished man. Body pale, rigor mortis present. Some hypostasis posteriorly, none anteriorly.

Glands.—Mass of enlarged glands along posterior border of sternomastoid. Hæmorrhagic on section, surrounding tissue infiltrated with blood-stained serous effusion.

Thorax.—The pericardium contained 2 ounces of turbid fluid. There was no pericarditis. Heart well developed, left side firm and contracted, right dilated and flabby, the large veins full of dark fluid blood. Left lung, no fluid in pleural cavity. Recent pleurisy, with adhesions the left base at back and front. The whole lung except the upper half of the upper lobe contained very numerous patches of gray granular consolidation, varying in size from 1 inch to $\frac{1}{16}$-inch in diameter. The upper half of the upper lobe was emphysematous, posteriorly, especially at the base of the lung, was very œdematous with reddish serous fluid. Right lung, no fluid in pleural cavity. Recent pleurisy, with adhesions over anterior border of upper lobe. The whole lung was filled with very numerous patches of grey consolidation, about $\frac{1}{2}$—$\frac{1}{16}$ inches in diameter, least numerous towards the apex. Posteriorly, especially at the base, the lung was œdematous with serous fluid. Bronchial glands enlarged, and black with hæmorrhage.

Abdomen.—Peritoneum normal. Stomach and intestines pale and distended with gas. Spleen decidedly large, deep purplish colour, and very soft. Liver somewhat large, congested, and a little tough. Kidneys pale, some cortical hæmorrhage, cortex narrow, contained a few yellowish necrotic patches, about $\frac{1}{8}$-inch in diameter. Capsule somewhat adherent, and substance toughened. Mesenteric glands enlarged and pinkish on section.

Bacteriological Examination.—

The affected glands, spleen, liver, kidneys, and broncho-pneumonic patches yielded pure cultures of *B. pestis.*

Case No. 110.—Age 27 years, male. Admitted 11th May, 1902, on the 1st day of disease. Temperature on admission 104·8, pulse 112, respirations 32. Onset, sudden. Glands affected, left parotid lymphatic gland, right and left submaxillary glands. He had 160 c.c. curative serum intravenously and

240 c.c. subcutaneously. Duration of acute symptoms, 3 days. Prominent feature, delirium. This case was complicated by an attack of facial erysipelas. He was discharged on 7th June, 1902.

Case No. 114.—Age 38 years, male. Admitted 26th May, 1902, on the 5th day of disease. Temperature on admission 103, pulse 106, respirations 38. Onset, sudden. Gland affected, left femoral. He had 460 c.c. curative serum intravenously. ¼ albumen was present. Prominent feature was marked heart failure and death 10 hours after admission, at 1·45 a.m. 27th May, 1902.

Post Mortem Notes.—

P.M. 27th May, 1902, at 10 a.m.. Well-developed, well-nourished body. Rigor mortis present and well marked. Some posterior hypostasis, but none anteriorly. There was an enlarged gland in the left femoral region, and one in the left inguinal region, the skin over the glands was hæmorrhagic, and the surrounding tissue was infiltrated with bloody serum. On section the glands were dotted with numerous hæmorrhages.

Thorax.—The pericardium contained about 2 ounces of clear fluid. The heart was well developed, the left side contracted and firm walled, right dilated, and somewhat pale and flabby. The pleural cavities were normal. Lungs anteriorly were pale, posteriorly there was marked hypostatic engorgement. On section the lungs were dark, almost black, and œdematous. The bronchial glands were tough and black, probably due to anthracosis.

Abdomen.—The peritoneum and intestines were normal. The spleen was enlarged, dark-purple in colour, and somewhat soft. The liver was venously congested, and rather tough. The kidneys were congested, capsule was easily stripped, substance a little tough. There was some subcapsular hæmorrhage in the left kidney. The pelves and calyces of both kidneys were filled with dark clotted blood. The mesenteric glands were not enlarged.

Bacteriological Examination.—

The spleen, liver, kidneys, and affected glands yielded pure cultures of typical *B. pestis.*

1903.

[Returned by Dr. J. E. F. McDonald.]

During this period nineteen cases were reported and came under observation; of these, nine were fatal. Two of these latter, however, were only discovered *post mortem.* All the cases were confirmed bacteriologically. In most, cultural and inoculation experiments were performed in addition to microscopic examination.

All the cases presented definite buboes, there being none of the so-called primary pneumonic or septicæmic cases.

In the majority the primary bubo was in the femoral region; it was frequently accompanied by smaller glands in the inguinal region of the same or opposite side. The primary bubo usually appeared on or about the second day of disease.

In two cases local lesions, in which the presence of *B. pestis* was demonstrated, were present in the drainage area of the affected glands.

The onset in the majority of the cases was very sudden, the first symptoms being commonly vomiting and frontal headache; a rigor or some shivering was frequently noted early in the attack.

Case No. 10.—An interesting case was that of an employee of the Health Department engaged in handling rats.

On a Tuesday, while engaged manipulating rats at the Bacteriological Institute, this man stabbed his hand with a pair of scissors. The wound was cleansed with 5 per cent. carbolic lotion, and healed rapidly. On the Thursday following (on which day one infected rat was found), while chopping wood at the rat cremation furnace, he was struck on the upper arm by a piece of wood, which abraded the skin. On Thursday, Friday, and Saturday he complained of being "seedy," and ate very little. On Sunday he felt "very ill," and his arm around the sore felt hot and painful; during the day this pain and soreness spread to the axilla.

He was seen by the Commissioner of Public Health that evening, and the wound was treated with pure carbolic acid crystals.

Neither then nor the next morning when he was examined by Dr. McDonald were his symptoms considered very suggestive of plague. On the Tuesday when examined he was much improved, but that evening he had a severe rigor, and next day (Wednesday) clinical and bacterioscopic evidence confirmed the case as one of pestis. On several occasions bacilli were found in the local sore, to which the pure carbolic was applied on Sunday, and not once in the enlarged tender glands in the axilla.

Pulmonary Complications.—Two cases came under observation. The first (14) was apparently recovering from pestis, and had reached the 8th day of his attack, with temperature falling, and respirations never above 30, and entire absence of abnormal physical signs in the chest when he suddenly presented symptoms of lobar pneumonia about midday, and was dead by 6 a.m. next morning.

The second case (15) admitted, with temperature 102, pulse 90, respirations 26; spat up blood-stained mucus, which contained a pure culture of *Bacillus pestis* (guinea-pig died in four days) on his second day in hospital, developed a cough next day, a "patch" of broncho-pneumonia on the fourth day, and died on the 14th day.

The first case was lobar pneumococcal pneumonia, confirmed by clinical, bacteriological, and *post-mortem* examinations.

The second differed in every respect, and was a lobular pestal broncho-pneumonia.

Both cases developed swollen glands in the neck.

Sudden Fatal Heart Failure.—This case was admitted with temperature 104, pulse 160, respirations 40, responded well to 200 c.c. serum given intravenously, the temperature dropping to 100 degrees next day, with pulse 140, respirations 32. At 7 a.m. next morning his pulse presented no change to either of his two nurses who examined it, yet at 7·15 a.m. he suddenly paled and gasped, and within a minute or two his heart had completely stopped. This case of sudden death, when a most hopeful prognosis seemed quite justifiable, and the sudden oncoming of fatal lobar pneumonia in Case 14 when, in both cases, the patient was apparently safe so far as any further constitutional effects of pestis were concerned, demonstrate the wisdom of a very cautious prognosis until the temperature has been normal for some days.

Heart Lesions Persisting.—Of the twelve cases discharged from the Plague Hospital, three of the female patients (æt. 12-20) had persistent slight arrhythmia and soft systolic bruit at all areas. In Case 7 the sounds were not even fairly clear until the ninth week, and in Cases 8 and 7 the systolic bruit persisted for sixteen and seven weeks respectively.

1904.

[Returned by Dr. C. C. Baxter-Tyrie.]

CASE 1.—Saddler. Infected rats discovered on premises during the preceding week. Initial temperature 103 degrees Fahr.; pulse extremely weak and dicrotic; serum 120 c.c. injected subcutaneously, 2nd day; temperature normal 4th day; right upper femoral gland affected; suppuration delayed and limited.

CASE 2.—Schoolboy. Plague next door last year; infected rats from store immediately adjoining; initial temperature 103 degrees Fahr.; pulse weak and dicrotic. Serum 200 c.c. subcutaneously. Temperature normal 10th day; had serum rash. Left upper femoral gland affected.

CASE 3.—Produce hand. Ill twelve days before coming under observation. Initial temperature 100 degrees Fahr.; dicrotic pulse. Temperature normal 7th day; gland resolved without suppuration. No serum was given. Left upper femoral gland affected.

CASE 4.—Produce hand (female). A bag-maker. Initial temperature 104 degrees Fahr.; dicrotic weak pulse. Serum 120 c.c. subcutaneously. Temperature normal 18th day; left upper femoral gland affected and sloughed entirely.

CASE 5.—Schoolgirl (from infected area). Initial temperature 105 degrees Fahr.; pulse 180, weak and dicrotic. Serum 240 c.c. subcutaneously, 120 c.c. intravenously. Temperature normal 17th day; upper left femoral gland affected. Very severe case.

CASE 6.—Produce bag-merchant. Initial temperature 105 degrees Fahr. Serum 240 c.c. subcutaneously. Temperature normal 5th day. Left upper femoral gland affected.

CASE 7.—Produce hand. Initial temperature 104 degrees Fahr. Serum 240 c.c. subcutaneously. Left upper femoral gland affected; little suppuration. Temperature normal 4th day. 17 days in hospital, the shortest period of detention of the cases.

CASE 8.—Produce hand. Initial temperature 104 degrees Fahr.; pulse extremely weak. Serum 200 c.c. subcutaneously. Died on the 2nd day. Complete renal suppression from time of admission. History of previous renal trouble. Autopsy findings: Chronic interstitial nephritis and general infection of the organs with the bacilli. Cultures of *B. pestis* were obtained from all the thoracic and abdominal viscera.

CASE 9.—Stable hand. Initial temperature 104·6 degrees Fahr. Obtained horse feed from infected store. Serum 200 c.c. subcutaneously; temperature normal 5th day. Left upper femoral gland affected; was not removed to Colmslie.

CASE 10.—Produce hand. Initial temperature 99·2 degrees Fahr. Serum injected subcutaneously. Died on the second day. General blood infection; bacilli were mixed in type, but mainly coccal.

CASE 11.—Grocer and produce hand (Chinaman). Initial temperature 104 degrees Fahr. Died immediately after being seen; had been ill 4 days. Large bubo implicating the whole left femoral group.

CASE 12.—Produce hand. Initial temperature 103·6 degrees Fahr.; pulse weak and dicrotic. Serum 102 c.c. subcutaneously. Temperature normal 5th day. Left upper femoral gland affected and resolved without suppuration.

CASE 13.—Grocer and produce hand. Initial temperature 104·6 degrees Fahr.; pulse 120, weak and dicrotic. Serum 320 c.c. subcutaneously. Left upper femoral gland affected; serum rash. Temperature normal 20th day. Gland sloughed *en masse*. Very severe case.

CASE 14.—Racehorse trainer. Produce obtained from infected store. Rats dying in numbers prior to his seizure. Initial temperature 105 degrees Fahr. Pulse dicrotic. Serum 240 c.c. subcutaneously, right upper femoral gland affected. Temperature normal 12th day.

CASE 15.—Oyster-saloon cook. Infected rats from the shop this year. Four days ill before seen; initial temperature 105·4 degrees Fahr. Pulse 152, dicrotic. Serum 120 c.c. subcutaneously. Died shortly after being seen. General blood infection.

CASE 16.—Produce hand. Initial temperature 102·6 degrees Fahr., pulse weak and dicrotic. Serum 120 c.c. subcutaneously. Temperature normal 6th day. Right upper femoral gland was affected and resolved without suppuration.

CASE 17.—*See* "Plague Lesions," page 64.

CASE 18.—Sanitary wharfman. Ill three days; died before arrival of Health Officer. Left upper femoral gland affected. Autopsy: General blood infection and secondary specific pneumonia.

CASE 19.—Estate agent. Initial temperature 104·8 degrees Fahr., pulse 104, very weak and dicrotic; left upper femoral gland affected. Serum 240 c.c. subcutaneously. Temperature normal 7th day. Dead infected rat found in offices.

CASE 20.—Stableman. Initial temperature 102·6 degrees Fahr., pulse 100, weak and dicrotic. Serum 240 c.c. subcutaneously. Left upper femoral gland affected; temperature normal 5th day. Serum rash. Produce from infected store.

CASE 21.—Schoolboy. Initial temperature 105 degrees Fahr.; pulse 136, weak and dicrotic. Serum 240 c.c. subcutaneously. Right upper femoral gland affected; temperature normal 6th day. Infected rats at store close by, which he was in the habit of frequenting. Family cat also brought rats to the house from this store. (Cf. case. New Farm outbreak, *see* page 64.)

CASE 22.—*See* page 64.

CASE 23.—Draper. Infected rats obtained in the vicinity of the home. Initial temperature 102·8 degrees Fahr., pulse 96, weak and dicrotic. Serum 120 c.c. subcutaneously. Temperature normal 9th day. Left upper femoral gland affected.

CASE 24.—Baker. Infected rats from bakehouse. Serum 120 c.c. subcutaneously. Died 5th day. General hæmal infection. Developed meteorism the 2nd day, and lapsed into an adynamic condition which continued until death.

CASE 25.—*See* "Plague Lesions," page 64.

1905.

[Returned by Dr. F. W. Woolrabe.]

Case 2.—E. McG., male, aged 17, furniture storeman. Admitted 27th January, 1905; discharged 26th February, 1905. Transferred from General Hospital. Illness began with pains all over body for a week; on 24th January, 1905, he began to vomit and had a rigor. Pulse dicrotic and feeble. Serum 120 c.c., later 120 c.c. Urine showed albumen for four days. Right femoral bubo, which suppurated. Main symptoms: Vomiting, insomnia, delirium, and cardiac weakness.

Case 3.—S. W., male, aged 18, plumber. Transferred from General Hospital 30th January, 1905; died 31st January, 1905. Sudden onset, with vertigo after exposure to sun, later a rigor, followed by abdominal pain and vomiting, and pain all over body. Sore in left leg. Temperature 104·8, pulse rapid and dicrotic. Left upper femoral bubo. Serum 120 c.c. He failed rapidly. Main symptoms: Delirium and pain.

Case 4.—J. W., male, aged 18, grocer's hand. Admitted 5th February, 1905; discharged 9th March, 1905. Temperature 104·8. Sudden onset, with headache and pain in left groin, three days later vomiting. Left femoral bubo. Serum 160 c.c. No albumen. Severe epistaxis on 3rd day.

Case 5.—*See* "Plague Lesions," page 64.

Case 6.—Kathleen S., female, aged 25, housewife. Transferred from General Hospital 7th February, 1905; died 22nd February, 1905. Sudden onset, 5th February, 1905, with severe headache and persistent rigors, followed by vomiting; admitted to General Hospital 6th February, 1905. Left femoral bubo. Serum 120 c.c., later 120 c.c. A little albumen. The patient reacted to the first dose of serum almost at once, but the improvement did not last. She gradually became weaker, and all her symptoms became intensified. Her mind was clear almost till her death. Main symptoms: Headache, vomiting, and persistent diarrhœa and hyperpyrexia. Extensive and brawny periadenitis.

Case 7.—J. J. S., male, aged 39, dairy farmer. Admitted 8th February, 1905; discharged 8th March, 1905. Sudden onset 1st February, 1905, with rigor, vertigo, and headache and fever. Illness increased, with vomiting and severe pain in left groin. Had 120 c.c. serum 2nd February, 1905. Had left femoral bubo, which suppurated. Was too ill for removal for some days. Main symptoms: Retention, meteorism, delirium.

Case 8.—R. P. T., male, aged 28, butcher. Admitted 12th February, 1905; discharged 18th March, 1905. Sudden onset 6th February, 1905, with rigor, and sensation of heat and nausea. Had to leave work next day with headache, vomiting, and vertigo. On 9th noticed painful left femoral bubo, which suppurated later. Very restless and thirsty, with temperature of 103·8, and a trace of albumen and dicrotic pulse on admission. Serum 120 c.c. Recovery uninterrupted.

Case 9.—H. G., male, aged 9, schoolboy, transferred from Children's Hospital 14th February, 1905; died 18th February, 1905. Sudden onset 18th February, 1905, with headache, vomiting, fever, and painful left femoral bubo. Serum 80 c.c., later 80 c.c. Delirious on admission, very restless, vomiting, coughed a good deal (no *B. pestis* in sputum). Condition grew gradually worse, with involuntary urination and abdominal pain. Main symptoms: Increasing cardiac weakness and continuous hyperpyrexia.

Case 10.—A. K., female, aged 19, domestic duties at home adjoining bakery. Admitted 16th February, 1905; died 27th February, 1905. History uncertain. Hyperpyrexia and dicrotism when seen. Right femoral bubo. Serum 140 c.c., later 120 c.c., but did not react to it. Mind was disturbed from the first, though she answered sensibly when spoken to; at other times she rambled. Later, meteorism developed, and her symptoms gradually increased, with insomnia, restlessness, and delirium. The cervical glands became affected. Meteorism became excessive, with diarrhœa and involuntary urination and continuous delirium. Main symptoms: Delirium, meteorism, implication of cervical glands.

Case 12.—Sarah P., female, married, aged 47. Admitted 16th February, 1905. History of eight days' illness beginning with fever, headache, and pains all over body. On 4th day pain centred in left groin. As she was recovering when seen, no serum was given. Admitted with large left inguinal and femoral buboes, which suppurated. Pulse dicrotic. Main symptom was her very lethargic and depressed condition. Recovery was complicated by a slight pleurisy.

Case 13.—M. S., female, aged 35, married woman. Admitted 17th February, 1905; discharged 11th March, 1905. Sudden onset 13th, with general pains, fever, and severe headache and pain in left groin. Serum 120 c.c. Left inguinal bubo, which suppurated later. Recovery complicated by attack of dengue.

Case 14.—E. R., male, aged 12, schoolboy. Admitted 19th February, 1905; discharged 15th March, 1905. Onset followed a cold of two weeks' duration, with pain in back and headache. Noticed pain in left groin 14th February, 1905, and swelling next day and day after. Vomited on 4th day. Temperature during these days rose to 105, but had fallen to 101 on 18th February, 1905. Left inguinal bubo, which suppurated. No serum. Recovery uneventful.

Case 17.—A. O'B., male, aged 19, cowboy. Admitted 9th March, 1905; discharged 25th March, 1905. Had noticed soreness of left femoral region for seven days, with headaches for three days, and swelling for one. Restless and delirious on night of 8th. Serum 80 c.c. on 8th, 80 c.c. on 9th. Left femoral bubo, which suppurated. A good deal of albumen. A pustule formed on right foot on 9th. Pulse dicrotic on admission. Had much meteorism the first three days, and profuse perspiration. Condition gradually improved. Main symptoms: Meterorism.

Case 18.—W. A. F., male, aged 25, boot hand. Admitted 4th April, 1905; discharged 21st April, 1905. On 28th March, 1905, noticed pain and swelling in left femoral region at night. Went to work on 29th, but had to leave with headache, vomiting, and pain in gland. Was delirious that night, with rigor. Further vomiting and rigor next day. On the 31st was delirious, with a rigor at night. Had

80 c.c. serum. Free perspiration and moderate delirium continued off and on till admission, with cough and frothy expectoration. Pulse at the same time very feeble. Had no albumen. Recovery after admission uneventful. Bubo left femoral, which suppurated. Main symptoms: Rigors and threatened cardiac failure.

Case 19.—F. J. L., male, 24, carpenter; had worked at produce store. Admitted 7th May, 1905; discharged 3rd June, 1905. Sudden onset, with pain in abdomen and left femoral gland, fever, and very severe headache. 100 c.c. anti-serum. Temperature declined till serum rash on 6th day of illness, but sleeplessness and great pain in bubo persisted for some days. Bubo suppurated. His recovery was complicated by a severe gonococcal polyarthritis with exacerbations of fever to 105·2. Main symptoms: Abdominal pain and headache.

Case 20.—I. F., male, 25 years. Admitted 7th May, 1905; discharged 2nd June, 1905 (accountant in produce store). Onset sudden, with headache and lumbar pains, and pain in right femoral gland; little fever. Serum 80 c.c. Bubo suppurated later. Trace of albumen.

Case 21.—G. P., male, 40 years, painter, lived next door to store. Admitted 7th May, 1905; discharged 3rd June, 1905. Sudden onset, with headache, vomiting, and pain in right femoral gland, and fever. Serum 40 c.c., later 100 c.c. subcutaneously. Gland suppurated. Main symptoms: Pain in bubo.

Case 22.—C. L. C., male, 38 years, plumber (got feed at store). Admitted 7th May, 1905; discharged 26th May, 1905. Sudden onset, with fever, headache, vomiting, pain in abdomen and left femoral gland. Had 40 c.c. of curative serum. Temperature steadily decreased. Had retention of urine, with trace of albumen. Main symptoms: Pain in abdomen and gland. Bubo suppurated.

Case 23.—W. B., male, 38 years, produce store keeper. Admitted 7th May, 1905; discharged 2nd August, 1905. Sudden onset, with fever, headache, pain in left femoral gland and abdomen, and vomiting, later epistaxis. Anti-serum 80 c.c., later 100 c.c. subcutaneously. Temperature steadily declined. Bubo suppurated. Main symptoms: Pain in abdomen and gland.

Case 24.—M.B., female, aged 30. Admitted 9th May, 1905; discharged 31st May, 1905; wife of A. B. Sudden onset, with fever, pain in left femoral gland and back of head. Had serum 60 c.c. subcutaneously. Uninterrupted recovery. Gland did not suppurate.

Case 25.—L. L. S., female, aged 31, married woman. Admitted 15th June, 1905; discharged 1st July, 1905. Sudden onset night of 7th June, 1905, with pain in groin, nausea, headache, rigors, no delirium, but some dreams. Had 80 c.c. serum 14th. Pulse very weak during that night. No albumen. After admission had some delirium and severe meteorism till 18th, with insomnia. Recovery uneventful. Left inguinal bubo, which suppurated. Main symptom: Meteorism.

1906.

[Returned by Dr. H. Chesson.]

Case No. 1.—Male, aged 25. Cellarman in warehouse at Petrie's Bight. Admitted 6th March. Temperature, 103 degrees Fahr.; pulse, 80; respirations, 32. Right femoral bubo. Onset 1st March. Had 200 c.c. Yersin's serum subcutaneously. Prominent features were: Marked cardiac depression, cyanosis, dicrotic pulse, and suppuration of bubo. Discharged 25th April.

Case No. 2.—Female, aged 27. Waitress at hotel in South Brisbane. Admitted 31st March. Temperature, 104·8 degrees Fahr.; pulse, 100; respirations, 28. Right inguinal bubo. Onset 23rd March. Had 100 c.c. Yersin's serum. Prominent feature was retention of urine. Discharged 28th April.

Case No. 3.—Male, aged 33. Rag merchant. Obtained rags from various places, also from rubbish tips. Admitted 14th April. Temperature, 103 degrees Fahr.; pulse, 100 (dicrotic); respirations, 28. Left femoral bubo. Onset 11th April. Ulcer on left buttock. Had 80 c.c. serum subcutaneously. Prominent features were: Slight retention of urine, suppuration of bubo. Discharged 28th April.

Case No. 4.—Male, aged 67. Fruit hawker. Obtained fruit from the markets, and frequented various produce stores. Admitted to the General Hospital on 13th April, in a dying condition. Temperature, 106 degrees; pulse not distinguishable at wrist, 150 at carotid; respirations, 40. Bubo in left axilla. Died shortly after admission. Specimens of spleen, axillary gland, lung and mesenteric glands all contained *B. pestis*, obtained *post mortem*.

Case No. 5.—Male, aged 16. Butter-packer, employed in warehouse in Mary street. Admitted 30th April. Temperature, 104; pulse, 112 (markedly dicrotic); respirations, 32. Small bubo of left axilla. Onset 26th April. Said to have cut a finger of his left hand with the edge of a tin while at work on 25th. Had 200 c.c. serum subcutaneously. Prominent features were: Restlessness, violence, and delirium. Markedly dicrotic pulse throughout. Died 3rd May.

Case No. 6.—Male, aged 15. Worked in a slaughter-yard. This case was reported by the police after *post mortem* examination on 16th May. Onset 13th May. Admitted to General Hospital on 15th. No bubo, but at the *post mortem* hæmorrhagic glands were found in groin, left pelvic region, mesentery, and neck. Spleen enlarged, and hæmorrhages in lungs.

Case No. 7.—Male, aged 15. Porter at Roma-street Station. Reported 8th June, but not moved. Temperature, normal; pulse, 76; respirations, normal. Onset 3rd June. Right inguinal bubo, in which *B. pestis* were demonstrated. An exceedingly mild case; convalescent within a week.

Case No. 8.—Male (Chinese), aged 30. Cook at hotel in Queen street. Admitted 20th June. Temperature, 104 degrees Fahr.; pulse, 100 (dicrotic); respirations, 32. Left axillary bubo. Onset 16th June. Had 200 c.c. serum. Prominent features: Delirium and loss of control of bladder and bowels. Died 21st June.

Case No. 9.—Male, aged 22. Woodcutter, Spring Hill dsitrict, city. Admitted to General Hospital 18th November. Temperature, 100·8 degrees; pulse, 118; respirations, 28. Right femoral bubo. Onset 18th November. Had 120 c.c. serum subcutaneously. Prominent features: Short course and sudden development of fatal symptoms, cyanosis and delirium, blood-stained sputum. Died 19th

November. *Post mortem:* Femoral, retroperitoneal thoracic glands enlarged and hæmorrhagic, liver and spleen enlarged and congested, lungs—both bases commencing consolidation, ecchymoses pericardial and peritoneal surfaces.

Case No. 10.—Male, aged 50. Locker in H.M. Customs. Admitted 23rd November. Temperature, 98·2 degrees Fahr.; pulse, 92; respirations, 24. Right inguinal bubo. Onset 16th November. Had 120 c.c. serum. Prominent features: This man had been about for a week ill, but not laid up. Suddenly developed serious symptoms, and died on 24th November.

Case No. 11.—Male, aged 21. Hairdresser in George street. Onset 15th December. Case reported. *Post mortem* on 18th December.

1907.

[Returned by Dr. H. Chesson.]

Case No. 1.—Male, aged 17. Office boy in warehouse in Queen street. Admitted 3rd January. Onset 31st December. Temperature, 104·2; pulse, 120 (dicrotic); respirations, 38. Bubo of left femoral gland. Specimens of lymph obtained from the bubo on 1st and 2nd January were all plague-negative, but one obtained on 3rd January revealed the presence of the *B. pestis.* A plague-infected rat had been found at his place of business a fortnight previous to his attack. Had 440 c.c. serum subcutaneously. Marked serum rash appeared on 7th day. Discharged 25th January.

Case No. 2.—Male, aged 39. Sailor, out of employment, seeking same round wharves. Admitted 14th January. Onset 11th January. Temperature, 104 degrees Fahr.; pulse, 108; respirations, 36. Bubo of right inguinal region. Suppuration of bubo. He had conjunctivitis and keratitis, which subsequently developed into a large suppurating corneal ulcer of the right eye. Had 120 c.c. serum. Discharged 15th April, after a prolonged convalescence.

Case No. 3.—Male, aged 14. Assisting in dairy. Admitted 16th January. Onset 15th January. Temperature, 104·6 degrees Fahr; pulse, 120; respirations, 32. Bubo of left femoral gland. Had 240 c.c. serum subcutaneously. Died 18th January.

Case No. 4.—Male, aged 12. Schoolboy, helping in store at New Farm. Admitted 18th January. Onset 16th January. Temperature, 103·8; pulse, 120 (dicrotic); respirations, 28. Bubo of left femoral gland. Had 200 c.c. of serum. Prominent features: The onset was marked by cerebro-spinal symptoms. This case, together with others following, occurring at the same time and coming from the same locality, being admitted to the Children's Hospital as cerebro-spinal meningitis; rapid and mild course. On the dorsum of left foot was a phlyctenule, from which *B. pestis* were obtained. Discharged 28th February.

Case No. 5.—Male, aged 14. Schoolboy, also assisting in his father's store at New Farm. Admitted 18th January. Temperature, 104; pulse, 128 (dicrotic); respirations, 28. Bubo of left femoral region. Had 360 c.c. of serum subcutaneously. Onset 14th January. Prominent features: In this case also the onset was marked by cerebro-spinal symptoms which, as in Case 4, caused a preliminary diagnosis of cerebro-spinal meningitis. Bubo suppurated. Discharged 2nd March.

Case No. 6.—Male, aged 13. Assisting at dairy. An associate of the two previous cases, and frequently in the produce store mentioned above. Admitted 18th January. Temperature, 105·6; pulse, 120 (dicrotic); respirations, 28. Bubo of left femoral region. Onset 16th January. Had 200 c.c. serum. The prominent features were: The development of cerebro-spinal symptoms at the onset—*e.g.*, headache, semi-unconsciousness, and delirium, general twitching and retraction of the head. Bubo suppurated. Discharged 2nd March.

Case No. 7.—Male, aged 10. Schoolboy, associating with the previous cases at the produce store in New Farm. Admitted 18th January. Onset 16th January. Temperature, 103·6 degrees Fahr.; pulse, 130 (dicrotic); respirations, 34. Bubo of right femoral region. Had 120 c.c. serum. This case was also admitted to the Children's Hospital with symptoms suggestive of cerebro-spinal meningitis. Bubo suppurated. Discharged 28th February.

Case No. 8.—Male, age 10. Schoolboy. Associated with four previous cases at the produce store in New Farm. Admitted 18th January from Children's Hospital with the previous four cases. Onset 16th January. Temperature, 103·6; pulse, 128 (slightly dicrotic); respirations, 28. No bubo, but one small, shotty gland in right groin. Had 120 c.c. serum subcutaneously. This boy had the same cerebral symptoms as the previous four cases, and, being closely associated with these, though having no bubo, and no bacteriological evidence being obtained, he was considered to be, and treated as, a case of pestis. Discharged 22nd February.

Case No. 9.—Male, age 13. Schoolboy, closely associated with the previous five cases. Admitted 18th January from the Children's Hospital. Onset 15th January. Temperature 102·4; pulse, 120; respirations, 24. No bubo. He had the cerebral and other symptoms, and in every way resembled the preceding case. No bacteriological evidence of plague obtainable. Discharged 22nd February.

Case No. 10.—Male, age 14. Schoolboy, living in same locality as the previous six cases, and going to produce store before mentioned for provisions. The back yard of his place of residence joined the back yard of store. Admitted 20th January. Onset, 14th January. Temperature, 103·8; pulse, 120; respirations, 32. Bubo of cervical gland first noticed on 21st January. Had 200 c.c. serum. Had sores on his lip and chin, but pus from these was negative. Died 25th February.

Case No. 11.—Female, age 75. Housework. The grandmother of Case No. 10. Lived in same house, and nursed him prior to his removal to Plague Hospital. She also visited the produce store. Case attended at home; too ill for removal. Temperature, 104·8; pulse, 80 (dicrotic); respirations, 34. Onset 19th January. Left femoral bubo. Had 120 c.c. serum subcutaneously. Prominent feature was rapid collapse. Died 21st January.

Case No. 12.—Male, age 8. Schoolboy, living in house the yard of which adjoined that of the store in New Farm mentioned above. Diagnosed *post mortem* on 21st January. Onset 18th January. Bubo of left femoral region. P.M. Hæmorrhagic glands of left femoral chain extending to iliac chain. Hæmorrhagic patches behind peritoneum. *B. pestis* numerous in specimens.

Case No. 13.—Male, age 8. Schoolboy, nephew of the owner of produce store at New Farm. Admitted 20th January. Onset 18th January. Temperature, 103·8; pulse, 132; respirations, 56. Bubo of left femoral gland. Had 200 c.c. Yersin's serum subcutaneously. Prominent features were: Vomiting during the first two days, and slight delirium. Discharged 2nd March.

Case No. 14.—Female, age 14. Housework. Sister of Cases 6 and 7, and living opposite the store at New Farm, where she frequently went for provisions. Admitted 21st January. Onset 20th January. Temperature, 105 degrees; pulse, 136; respirations, 30. Bubo of left femoral gland. Had 360 c.c. Yersin's serum subcutaneously. Prominent features were: Incontinence of urine and suppuration of gland. Discharged 2nd March.

Case No. 15.—Female, age 9. Schoolgirl, daughter of owner, and living at produce store, New Farm. Admitted 23rd January. Onset 22nd January. Temperature, 104; pulse, 120 (dicrotic); respirations, 40. Bubo of left femoral region. Had 360 c.c. serum subcutaneously. She was extremely sleepy at first, and afterwards delirious. Gland suppurated. Discharged 9th March.

Case No. 16.—Male, age 40. Labourer, working at produce store, New Farm, father of Cases 6, 7 and 14. Admitted 23rd January. Temperature, 104·4; pulse, 120; respirations, 30. Buboes of left axilla. Onset 22nd January. Had 320 c.c. serum subcutaneously. Had a phlyctenule on the flexor surface of left forearm, matter from which contained *B. pestis.* Developed pneumonic symptoms, with presence of *B. pestis* in sputum on the 8th day. Died 30th January.

Case No. 17.—Male, age 49. Health Department gang working at the produce store in New Farm which was the apparent centre of infection of the preceding Cases Nos. 4 to 16. Admitted 26th January. Onset 24th January. Temperature, 104 degrees Fahr.; pulse, 104 (dicrotic); respirations, 30. Bubo of right femoral gland. Had 740 c.c. Yersin's serum. On the 8th day this case developed pneumonic symptoms, with *B. pestis* in his sputum. His bubo also gave a lot of trouble; it was twice incised, and eventually after his final discharge from the Plague Hospital a mass of glands had to be dissected out. Discharged on 22nd March, but was not fit for work until June.

Case No. 18.—Male, age 42. Employed on Health Department gang working at above-mentioned store in New Farm. Admitted 26th January. Onset 24th January. Temperature, 103; pulse, 112; respirations, 28. Bubo of left inguinal gland. Had 240 c.c. serum subcutaneously. Developed pneumonic symptoms, with *B. pestis* in sputum. Died 31st January.

Case No. 19.—Male, age 52. Employed on Health Department gang working at above-mentioned store in New Farm with the two previous cases. Admitted 29th January. Onset 26th January. Temperature, 103·8; pulse, 80 (intermittent); respirations, 24. Bubo of left femoral region. Had 280 c.c. serum subcutaneously. Most prominent feature was delirium, with at times violence. Discharged 5th March.

Case No. 20.—Female, age 58. Assisting husband in painter's shop in city. Admitted 2nd February. Onset 30th January. Temperature, 103; pulse, 120 (dicrotic); respirations, 30. Buboes of right and left femoral glands. Had 240 c.c. serum subcutaneously. Rapidly developed heart failure. Suppression of urine. Died 5th February.

Case No. 21.—Male, age 20. Driver of baker's cart. Admitted 1st February. Onset 31st January. Temperature, 103; pulse, 120; respirations, 36. Bubo of left femoral region. Had 520 c.c. serum subcutaneously. Prominent features were: Retention of urine, development of pneumonic symptoms, with *B. pestis* in sputum. Died 9th February.

Case No. 22.—Male, age 50. Carter for city warehouse. Admitted 6th February. Onset 4th February. Temperature, 102·6; pulse, 94; respirations, 30. Bubo of right inguinal region. Had 230 c.c. serum subcutaneously. Discharged 5th March.

Case No. 23.—Male, age 19. Crockery packer in city warehouse. Admitted 9th February. Onset 7th February. Temperature, 102 degrees Fahr.; pulse, 112 (dicrotic); respirations, 26. Bubo of left femoral gland. Had 260 c.c. serum. Mild case; no complications. Discharged 5th March.

Case No. 24.—Female, age 46. Cook and laundress in hotel in city. Diagnosed *post mortem* on 22nd February. Onset 18th February. P.M. Spleen enlarged and softened. Deep iliac gland on both sides hæmorrhagic and slightly enlarged, the tissues surrounding them soddened with hæmorrhagic effusion. Femoral and inguinal glands injected, but not enlarged. *B. pestis* present in specimens.

Case No. 25.—Male, age 45. Squatter, staying at hotel in city. Admitted 7th March. Onset 5th March. Temperature, 104 degrees Fahr.; pulse, 100 (dicrotic); respirations, 26. Bubo of right femoral region. Had 400 c.c. of serum. Severe, but uncomplicated, case. Discharged 13th April.

Case No. 26.—Male, age 23. Collar-maker at saddler's shop in city. Admitted 11th March. Onset 10th March. Temperature, 104 degrees Fahr.; pulse, 120 (dicrotic); respirations, 30. Bubo of left femoral region. Had 480 c.c. serum. Prominent features: Blood-stained sputum, containing *B. pestis;* no other signs of chest complication. Bubo suppurated. Discharged 4th May.

Case No. 27.—Male, age 48. Sculleryman at hotel in city. Admitted 17th March. Onset 14th March. Temperature, 104 degrees Fahr.; pulse, 120; respirations, 36. Bubo of right femoral gland. Had 200 c.c. serum. Gland suppurated. No complications. Discharged 20th April.

Case No. 28.—Female, age 23. Household duties. She was admitted to the Lady Bown Hospital, and was confined of full-term child on 5th April. On 7th April she had a bubo of left femoral gland. Temperature, 105; pulse, 132; respirations, 26. She had 160 c.c. serum. Died at the Lady Bowen Hospital, 8th April.

Case No. 29.—Female, age 45. Housewife. Admitted 10th April. Onset 7th April. Temperature, 101 degrees Fahr.; pulse, 94; respirations, 22. Bubo of right femoral gland. Had 160 c.c. serum subcutaneously. Bubo suppurated. Mild case. Discharged 4th May.

Case No. 30.—Female, age 14. Household duties, living at home. Admitted 10th April. Onset 9th April. Temperature, 104 degrees Fahr.; pulse, 108; respirations, 24. Bubo of left femoral gland. Had 380 c.c. serum. Prominent features were delirium and development of bubo in neck. Died 17th April.

Case No. 31.—Male infant, age 7 days. The child of Case No. 28. This child was born in the hospital and was removed from its mother as soon as she was recognised as suffering from pestis. He had been placed twice to the breast previously. Was given a small prophylactic dose, 4 c.c., of Yersin's serum. On 10th April (4th day after birth) temperature rose to 101 degrees, and the next day to 103 degrees. No bubonic swellings palpable. Died on 12th April. Specimens obtained *post mortem* revealed the presence of *B. pestis.*

Case No. 32.—Male, age 12. Schoolboy and flower-seller. Diagnosed *post mortem* on 20th May. Onset 18th May. Temperature, 104 degrees; pulse, rapid; respirations, rapid. Bubo of right submaxillary gland. Specimens obtained at P.M. revealed presence of *B. pestis.*

Case No. 33.—Male, aged 18. Cook at hotel in city. Admitted 25th May. Onset 23rd May. Temperature, 105; pulse, 122 (dicrotic); respirations, 40. Bubo of left inguinal gland. Had 240 c.c. Yersin's serum subcutaneously. Prominent features: Restlessness and delirium, suppuration of gland. Discharged 29th June.

Case No. 34.—Male, age 17. Yard and kitchen boy at hotel in city. Worked with previous case at the same hotel. Admitted 29th May. Onset 26th May. Temperature, 105·4; pulse, 132; respirations, 30. Bubo of right femoral gland. Had 320 c.c. serum subcutaneously. Gland suppurated early. Discharged 29th June.

Case No. 35.—Male, age 45. Licensee of hotel where the two previous cases worked. Seen by Acting Health Officer on 31st May. Onset 26th May. Temperature, normal; pulse, 80. Bubo of left femoral gland, suppurating. *B. pestis* found in specimen after inoculation experiments. A very mild case; convalescent when seen. No serum given.

Case No. 36.—Male, age 39. Telegraph operator. Seen by Health Officer 1st June. Onset 31st May. Temperature, 103·8 degrees Fahr.; pulse, 120 (dicrotic); respirations, 30. Bubo of left femoral gland. Had 120 c.c. serum subcutaneously. Died suddenly before he could be removed to Plague Hospital, on 1st June.

IV.—CLINICAL BACTERIOLOGY.

In the living patient material for bacteriological examination was obtained from the interior of the bubo by puncture with a sterilised hypodermic syringe. Before the stage of suppuration was reached the bubo was found to contain plague bacilli in a greater or less number. These bacilli on bacterioscopic examination were proved to be non-motile, gram-negative, bi-polar organisms. In fatal bubonic and septicæmic cases the organism was found widely distributed in the spleen, lungs, liver, kidneys, heart-blood, and lymphatic glands. In pneumonic cases the plague bacilli were demonstrated in the sputa.

With a view to determining the vitality of the *B. pestis* in pus, certain experiments were carried out by Mr. Pound, the Government Bacteriologist, in 1904. The results obtained showed that, with two exceptions, pus containing plague bacilli from suppurating glands of infected guinea-pigs, when inoculated subcutaneously into healthy guinea-pigs, caused the death of the latter in from four to six days; *post-mortem* examination of the bodies revealing the presence of large numbers of plague bacilli in various internal organs. In every case the wounds from which the pus was obtained had been in a suppurating condition for more than five days, and in two instances the pus had been discharging for fourteen days.

It was not common to find plague bacilli in the peripheral blood of bubonic cases, except at a period immediately prior to the death of the patient; but in septicæmic cases the plague organism was frequently demonstrated in blood-films prepared at a fairly early stage of the disease.

In several cases plague bacilli were found in enormous numbers in the peripheral blood without producing any severe constitutional disturbance, the clinical symptoms being comparatively trivial until just prior to death. (*See* page 64.)

The lung tissue would appear to have a special power of exalting the virulence of the *B. pestis;* guinea-pigs inoculated with material from the lungs of an animal dead of plague, or with the sputum of a patient dying of primary plague pneumonia, succumbing in a much shorter time than other guinea-pigs inoculated with material taken from the spleen and other organs.

In all suspected cases of plague in the living subject, a "specimen" of juice from the bubo, blood, or sputum was obtained.

In *post-mortem* cases it was customary to examine portion of the spleen, lungs, liver, kidney, and lymphatic glands.

Bacteriological Diagnosis.—In every case, with but few unavoidable exceptions, the material was submitted to the three following tests:—

1. Microscopic examination of stained film-preparations.
2. Inoculation of the suspected material on culture media.
3. Subcutaneous inoculation of a guinea-pig.

The cases were not so many in number or so frequent in sequence during an epidemic in Brisbane as to prevent the above tests being strictly carried out.

The diagnosis of plague, therefore, in the great majority of the cases was thus readily placed beyond doubt by the bacteriological method devised.

The triple test—smear, culture, and animal inoculation—was positive in the majority of the cases diagnosed clinically as plague, but the results in mild cases varied as follows:—

Class.	Smear.	Culture.	Inoculation.
A	Positive	Positive	Positive
B	Negative	Positive	Positive
C	Positive	Negative	Positive
D	Negative	Negative	Positive.

The application of the bacteriological test to doubtful cases has already been mentioned in dealing with administrative measures, the time required for bacteriological proof often extending over several days in cases where animal inoculation, as in Class D, was the only positive evidence obtainable. Under this class may also be included those mild forms of plague commonly referred to as "*Pestis Minor.*"

Morphology.—The typical plague bacillus is a short thick rod, of an oval to cylindrical shape, rounded at both ends and presenting, when stained, a resemblance to a small shut safety-pin.

The *B. pestis* exhibits great variability in form and size. This pleomorphism must be constantly kept in view when forming an opinion as to the nature of a film preparation. Before giving a definite pronouncement upon a human case clinically suspected to be plague, and especially in times outside those of an epidemic, it is desirable that the cultural characteristics and pathogenic properties of the organism in question be ascertained. The same applies to rats, the bacteriological diagnosis of plague in rats being no less important than the examination of human cases, for the reason that bi-polar organisms unconnected with plague have been found in rats suffering from one of the several forms of acute infectious diseases to which they are liable.

A dependence on the microscope alone may result in the serious error of bacteria morphologically identical with the *B. pestis* being mistaken for that organism.

Bi-polarity, although the most constant, is not the predominant feature of the plague bacillus.

The tendency of the organism to exhibit bi-polarity in material taken from the tissues of animals was not unfrequently lost in cultivation on laboratory media, the organism being stained uniformly. In the septicæmic type of plague, in rats, the bacilli were often found to be uniform in shape and size and evenly stained.

In guinea-pigs, on the other hand, the bacilli were often of varied shapes—large and round, oval and small, long and thick. "A peculiar feature," Mr. Pound states, "was that in some of the guinea-pigs inoculated with a pure culture of plague—in fact, a colony of *B. pestis*, all of one shape and size—after the death of the animal the organisms appeared different in the tissues of the various organs examined. For instance, in the lymphatic glands the bacilli were elongated and typically bi-polar, in the lungs they were round like cocci, while in the spleen and liver both varieties were found to be present."

In some cases of septicæmic plague in human beings, the material examined was found to contain extremely small organisms, like micro-cocci, and stained in a very irregular manner. On agar and blood serum, and on young cultures in broth, the bacilli were fairly uniform in size and shape; but in old cultures on agar involution forms were very common. It was also found that though the morphological characters of the bacilli were different in several cases coming from the same locality and occurring simultaneously in the epidemic, yet, after passing through a culture medium and a susceptible animal no difference in the appearance of the bacilli was found to exist.

In a few microscopic specimens from cases of plague in man the *B. pestis* presented the appearance of a diplo-bacillus or a diplo-coccus, surrounded by a mucoid capsule difficult to stain. In the great majority of film preparations of the bubo material the bacilli were abundant, easy to stain, and easy of recognition. It is claimed by some observers that the plague bacillus forms a characteristic appearance in bouillon, to which a drop or two of oil, fat, or ghee have been added. When incubation takes place at a temperature at which the ghee remains solid, its under surface becomes covered with "stalactites," the appearance known as "Haffkine's stalactite growth" being considered as characteristic for *B. pestis.* At the present time, in the Bacteriological Institute, Brisbane, there are several broth cultures of the *B. tuberculosis*, made by Mr. Pound, which show stalactite growths almost identical with those of plague cultures.

The Government Bacteriologist has furnished me with the following report on the staining methods adopted by him, together with some notes on the pleomorphism of the *B. pestis.* He states:—

In the preparation of stained specimens of serum or blood, it is advisable not to pass the recently dried smear through the flame, as is usually recommended in text-books. It is only reasonable to expect that, with such a delicate substance as the protoplasmic contents of a bacterial cell, the sudden change of heat and cold occasioned by passing a prepared cover-glass through the flame, there will be some material alterations in the structural and morphological character of the organism. It is a golden rule in this Institute never to adopt such a method, which is continually being copied from book to book.

The two following methods demonstrate that peculiar morphological character—the bi-polar appearance—in a most perfect manner:—

1. Place a droplet of infected blood or lymph near the edge of a clean cover-glass, and immediately smear the same across the glass with a small strip of freshly-torn filter paper. This has the effect of making several wide furrows in the film, where the bacilli and a few blood or lymph cells are spread in such a perfectly thin layer as to cause many of the bacilli to be in complete optical contact with the cover-glass. Allow the film to dry spontaneously. Then float on the surface of a small quantity of carbol-fuchsin, to which has been added a few drops of alcohol to remove all traces of that green metallic-looking scum, which adheres so tenaciously to all glass objects. The staining may be intensified considerably by gentle heating. After two minutes staining the cover-glass is washed in water, and then in a watch-glass of water containing a few drops of alcohol, which tends to clarify the specimen. Wash again well in water, dry in the air, and mount in Xylol balsam.

2. After the smear is dried, float, prepared side down, on a 5 per cent. carbolic solution for about ten minutes then transfer direct to ordinary methylene blue solution for another ten minutes or longer, according to the density required. The specimen is now washed in water, dried, and mounted in balsam.

Either of these methods shows typical bi-polar staining, the central portion of each bacillus being quite clear or only faintly stained, while the extremities or poles are very deeply stained, gradually shading off towards the centre, while the cell wall has a clear, sharply defined margin.

By the carbol-fuchsin method specimens will retain their brilliancy for about five years.

Gram's Stain.—It is generally conceded by most writers on plague, that the bacillus is not stained by Gram's method. This is somewhat incorrect, for, on many occasions, I have found the bacilli from human cases, as also from rats and guinea-pigs, stain readily and uniformly by this method.

Simpson states that they may not be decolourised if a 50 per cent. spirit is used instead of absolute alcohol. But I maintain that, not only does the latter not decolourise some forms, but that they are unaffected by immersion in oil of cloves, which is a far greater solvent of aniline dyes and clearing agent than absolute alcohol.

Plague bacilli, stained by Gram, always appear considerably smaller than when simply stained by methylene blue or fuchsin. This may be explained by the fact that only the protoplasmic contents of the cell take the pigment, leaving the cell wall unstained. Further, the fixing with the iodine-iodide of potassium causes a decided undue shrinkage of the cell contents. This feature is well brought out in staining anthrax bacilli, which, by the simple treatment with either methylene blue, fuchsin, gentian violet, or bismarck brown, appear as fairly thick rods with parallel sides and perfect square cut ends; but, when stained by Gram are much thinner, the sides having a tendency to be uneven and the extremities distinctly rounded.

After staining plague bacilli by Gram their shape is very much altered. The majority appear as diplo-cocci, others as short rods, and evenly stained. Very few indeed only show a tendency to bi-polar staining.

Sections of tissue containing plague bacilli with bi-polar appearances may be most satisfactorily shown by first of all treating the section with 5 per cent. carbolic, and then placing on a watch-glass containing a freshly-made watery solution of monochrome methylene blue for fifteen minutes, and then washing in water and alcohol, and finally clearing in xylol and mount in balsam.

Both Romanowsky and Jenner stains show the polarity of plague bacilli faintly, but clearly, and in contrast colour to the blood and lymph cells, but these methods are not to be compared with the rapidity and perfection of results of the carbol-fuchsin or methylene blue.

Pleomorphism.—It is a well-established fact that plague bacilli are the most pleomorphic of all pathogenic bacteria. The greatest variation is seen when the bacilli are cultivated on artificial media, particularly agar-agar, when the culture is several days old. On such a culture very few organisms are seen to appear as what might be termed normal, that is, short rods with parallel sides and rounded ends which are stained much deeper than the central portion.

The majority of organisms are large, and, while many have a tendency to be spherical, others are either wedged-shaped or elongated, with marked irregular sides. As a rule, all of these abnormal or involution forms do not stain deeply with the usual aniline dyes.

Generally, in a rat dead of septicæmic plague the bacilli take the stain readily, are more uniform in shape, and are more or less cylindrical; while in guinea-pigs affected with generalised plague it is usual for the organisms to have a tendency to be more or less spherical, but in some cases of plague in rat, guinea-pig, and also the human subject, there appears to be a general mixture of varieties of organisms. Certainly, in most plague cases, either human or animal, the bacilli are generally larger in the localised form than when the disease is generalised, and of an acute septicæmic character.

V.—CLINICAL PATHOLOGY.

The *post-mortem* appearances of plague are usually those of a septicæmia. Vascular dilatations and hæmorrhagic extravasations with œdema, sum up the more essential features. The above are the cause of the soft, friable, and matted condition of the tissues, the engorgement with blood of the substance of the lungs, liver, spleen, kidneys, &c., the petechiæ on the surface of the liver and in the pelvis and calices of the kidney, and under the skin.

The primary bubo, in the early stage of the disease, shows the usual signs of inflammation—congestion and cellular increase—which quickly extends to the periadenitic tissue, the extravasated blood and serum binding the affected gland and the surrounding tissues together.

The primary gland may consist of one gland, or a group of glands, the sero-sanguinolent effusion into the areolar tissue surrounding the bubo forming with the latter a tense, somewhat elastic, swelling.

The palpation of a plague-bubo before the gland has become lost in the effusion is, to an experienced observer, somewhat diagnostic of the diesase. There is a peculiar sensation conveyed to the sense of touch about a bubo, the result of the plague virus, that is distinctive from buboes formed through other causes. When gently grasped between the first two fingers and the thumb the impression conveyed is not easily forgotten, the consistence being something like that of the testicle, to which it has been aptly likened. In the septicæmic form of plague there is always enlargement of the internal glands. The enlargement and œdema, however, are not so pronounced as in the case of the primary bubo. The retro-peritoneal glands, and frequently those of the mesentery, are enlarged and hæmorrhagic. A large number of, if not all, groups of lymph-glands are affected, so as to form continuous chains; the groin glands with the pelvic and the groups surrounding the iliac vessels; the cervical with the bronchial and mediastinal, and so on. The most conspicuous and characteristic pathological changes are those associated with the lymphatic system, which is the *locus prædilectionis* for the development of the plague bacillus in the body. As in typhoid fever the enteric ulcer is mostly confined within the limits of the follicles or follicular patches of the intestines, so in plague the one characteristic sign is the inflammation and swelling of the internal lymphatic glands, a condition always present, even where the external glands are not notably affected. The constant involvement of the upper external or upper internal femoral gland as the gland initiating the primary bubo in the bubonic cases has already been remarked upon. This preponderance of groin buboes is quite in harmony with what has been found in epidemics of other countries.

The lesion is altogether too regularly frequent in its incidence to admit of explanation on the ground of mere coincidence.

The preponderance of left-sided buboes also requires more explanation than the one of mere chance. Evidence of the possibility of the infective material entering by a breach of the surface of the skin has been afforded in the particulars of the few cases already given where direct inoculation was effected by rat-bites, *post-mortem* wounds, and other accidental abrasions on the upper extremities caused axillary buboes. Phlyctenules and plague-sores developing at the site of inoculation were only found in a small percentage of cases. Confirmation that the infective material enters through the skin, other than by flea-bites, has also been sought in other directions. Comparison has been made by certain observers between the Chinese and Hindus who walk barefoot, and Europeans who do not adopt this practice. Although many of the school children of Queensland go barelegged and barefooted during the summer months, the working classes are as well shod as in other countries, and the incidence of groin buboes are no more frequent in the former than the latter.

The broad inference with regard to primary buboes, then, is only to the effect that the system is invaded for the most part from external rather than internal surfaces.

Secondary buboes may not be preceded by primary buboes. They originate when the circulation is invaded by the plague bacilli, the blood stream becoming the agent for the distribution of the bacilli to different organs and glands in the body. The disease then becomes septicæmic. There is reason to believe that infection of the blood takes place, as a rule, during the period of incubation, but that the *B. pestis* during this period passes out of the blood stream into the lymph channels, to be ultimately arrested in a particular gland or group of glands, most commonly the glands in the region of the groin. The destruction of the walls of the blood vessels, inside and outside the glands, leading to hæmorrhage and inflammatory exudations, and followed by necrotic and disintegrating processes appears chiefly to be brought about by the destructive action of the plague bacilli and the toxines which the latter produce. The necrosis is generally most marked in the central portions of the gland, the hæmorrhages and inflammatory processes, together with the plague bacilli, appearing at the periphery. In taking a "specimen" of bubo material, therefore, it is important that the hypodermic needle should be inserted in directions to reach both the centre and periphery of the bubo before the final withdrawal of the needle.

While secondary buboes may and frequently do develop in all regions of the body quite independently of the seat of a primary bubo, the latter, however, may occasionally develop after the onset of constitutional symptoms:—

Case No 38, age 47 years, female.—Died 3rd February, 1902. Case diagnosed p.m. Onset, sudden. Temperature. 102·6; pulse, 110; respirations, 30 first day, maximum 103·8. Duration of illness, four days. No serum. Femoral bubo found at death, which was sudden and unexpected.

Case No. 104, age 45 years, female.—Admitted to the General Hospital on the evening of the 5th May, 1902. Her temperature was 101, and pulse 120. Patient's daughter stated she was quite well until the 2nd May, when she had a shivering attack, headache, and vomiting. Her speech became very thick, and could not be understood, and she could not walk. During the night she was in the hospital a gland arose in her left cervical region. She had 120 c.c. curative serum intravenously on the morning of the 6th, but died about noon on that day.

Blood.—The results in connection with the examination of blood-films will be found at page 64. In one or two fatal septicæmic cases plague bacilli were found in large numbers in the peripheral blood without accompanying severe constitutional symptoms in the early stages of the disease.

Urine.—In one case, before death, and in one case after the death of the patient, plague bacilli were recovered from the urine by culture, and by animal inoculation with the sediment of the urine.

Sputum.—The exact diagnosis of the primary pneumonic form of plague depends upon a bacteriological examination of the sputa. In two cases the pneumo-coccus was associated with the plague bacillus, and in another case the strepto-coccus and staphylo-coccus were found with the plague organism.

VI.—INCUBATION PERIOD.

The incubation period of plague varies between one to five days. Cases have been recorded in which the period of incubation appears to have ranged from a few hours to twelve or more days; but the latter period is decidedly rare, and for all practical purposes may be disregarded. The extreme limit of the incubation period may be fixed at five days, and it is seldom longer. Five days is the limit of duration of detention for quarantine purposes proposed by the Paris Convention of 1903.

VII.—PROGNOSIS.

The prognosis in individual cases depended on the race of the person attacked, the variety of plague, the situation of the primary bubo, and the period of the epidemic when attacked.

Practically all the coloured patients succumbed. The pneumonic and septicæmic types of plague generally had a fatal termination.

In the bubonic type, the situation of the local adenitis appeared to influence the degree of constitutional symptoms, patients with cervical buboes having the highest mortality.

Axillary buboes also gave an unfavourable prognosis, while patients exhibiting multiple buboes, and with the presence of bacilli in the blood, denoting a general hæmal infection, had small chance of recovery.

It was also observed that where atypical and coccal forms of *B. pestis* were present in the film preparation, made from the juice of the affected gland, in addition to the ordinary or typical forms of the bacillus, the prognosis was much graver than in cases where the organisms conformed to the usual type.

Attacks towards the close of an epidemic were, as a rule, milder than at the beginning, when atypical symptoms frequently characterised the first few cases.

The period of suspense was usually only a few days, death commonly occurring on the 3rd, 4th, or 5th days. If the patient survived the first week, a favourable termination was looked for.

A guarded prognosis as to the future course of the illness should always be given, for, apart from the fact that plague exceeds all other infectious diseases in danger, cases of sudden death from heart failure were not infrequent even when a most hopeful prognosis seemed justified and the patient was apparently progressing rapidly towards convalescence.

VIII.—DIAGNOSIS.

In the majority of cases, during the existing epidemic, the diagnosis can be made without much difficulty if with the clinical symptoms, the usual bipolar, non-motile, gram-negative bacilli are found in the film preparation made.

The bacteriological demonstration for the detection of the plague bacillus in the bubo juice or other material taken from the tissues, and conforming to certain morphological, cultural, and pathological characteristics, has already been described under " Clinical Bacteriology." It may be said, however, that bacteriological diagnosis was not always an infallible test so far as the smear and culture preparations were concerned, especially in the milder types of plague. Though bacteriological proof, as far as possible, was insisted upon in all cases, and, as experience proved, was of primary importance, the great majority of bubonic cases were capable of being diagnosed by clinical methods alone.

Clinically, difficulty arose in cases of plague of so mild a nature that the real nature of the disease was never suspected, or so severe in attack that death ensued before any characteristic symptom had developed.

The presence of an external bubo, together with the usual constitutional symptoms during the course of an epidemic, left little room for doubt, but such recognition was not always so easy in certain groups of cases in which external buboes were not early manifested or were absent altogether. This group included the early cases in the New Farm outbreak, the symptoms simulating cerebro-pneumonia without buboes was mistaken for symptoms of ordinary broncho-spinal meningitis; as also in the Maryborough outbreak, where primary pulmonary pneumonia.

In every successive and recurring epidemic a large proportion of "suspected" cases were reported by medical men to the Department for diagnosis. The great majority of these cases were negatived after minute clinical and bacteriological investigation; the largest number of negative cases coming under notice during the months in which the largest number of positive cases were proved. The fact that all cases suspicious of plague are required under the Plague Regulations to be reported would account for the large percentage of suspected cases reported to the Department, and many cases were also referred, doubtless, to the Department from motives of prudence rather than of real doubt.

Cases of typhoid fever, pneumonia, meningitis, dengue fever, and filariasis were reported as plague, and, in one case of skin lesion, the plague sore was thought at first to be anthrax.

The *serum diagnosis* of plague by agglutination methods was found of little practical importance. It was hoped that, as a result of research by numerous observers on agglutination, and on "hanging-drop" clumping and inhibition of growth, it would be possible to demonstrate the presence of *pestis* in those cases in which it is difficult to demonstrate bacilli, particularly those presenting themselves late in the disease, or in very mild or minor cases. The few experiments carried out were inconclusive and unsatisfactory so far as the application of the method of serum diagnosis of doubtful cases, or discrimination of plague from other diseases, was concerned.

The following are examples of some of the more important cases which were referred for diagnosis:—

CASE 1.—*Filarial Fever:* A. G., female, aged 18 years, complained of vomiting, headache, lassitude, and pyrexia. Her temperature, when seen by a medical man, was 102·5, pulse 112, respirations 32. There was a lymphangitis of the left arm and a bubo in the left groin. This latter swelling proved to be a lymphatic varix. The onset was sudden, and the clinical symptoms resembled plague. Dead embryo filariæ were detected in the blood.

CASE 2.—*Pneumo-coccal Infection:* A. McD., male, aged 16 years, office boy, complained of pain in left leg and thigh. Had a rigor. Left femoral gland enlarged. Temperature 104·2, pulse 92, respirations 30. There was a lymphangitis extending from the heel to the femoral gland, but no lesion was found on the heel. The bubo was pierced with a hypodermic needle and some serum withdrawn, which, on bacterioscopic examination was shown to contain pneumo-cocci. Slight cough and expectoration. No *B. pestis* or pneumo-cocci were found in the sputum; 100 c.c. of Yersin's serum was injected subcutaneously. The patient died eight days after onset of illness.

Autopsy: The lungs were found inflamed, and all the lymphatic glands were enlarged and hæmorrhagic, pneumo-cocci being found in considerable numbers. No *B. pestis* were discovered.

CASE 3.—*Anthrax:* See page 64.

CASE 4.—*Streptococcal Infection:* W. M., male, aged 19 years, complained of pain in right femoral region. The right femoral glands were enlarged, and the part was hot, red, œdematous, and painful. Temperature 102, pulse 90, respirations 20. The bubo rapidly increased in size, and suppurated. Clinically, the case resembled plague, and 100 c.c. of curative serum was injected. Streptococcus pyogenes aureus was detected in the pus, but the most minute examination failed to reveal the presence of the *B. pestis.* The patient made a good recovery.

CASE 5.—**Septicæmia. Heart Failure:* A. K. was admitted to the Brisbane Hospital about seven o'clock on the evening of the 28th May, 1907. She stated that she was quite well until three days before, when she began vomiting almost continuously. The vomited matter was at first a green fluid, which was gradually becoming whiter. She complained of pain in the right side of her chest and the epigastrium. Her temperature was 98, pulse 120, and respirations 28, on admission. There was some slight tenderness on the right side of the abdomen. The pulse was weak, and inclined to be dicrotic, and, after consultation, she was prepared for operation, when she began to vomit a dark-brown fluid. She was operated on about midnight, and the appendix was found to contain ten seeds on removal. There was no inflammation in or around it, and nothing could be found to account for her condition. She was removed back to the ward with a pulse of about 130, getting weaker and more irregular. She gradually got more restless, her pulse becoming thready and imperceptible by half-past five, and she died at ten minutes past six in the morning.

Permission was given to hold a *post-mortem* examination. The body was rapidly turning black within four or five hours after death. A small gland could be felt in the right groin; no other glands could be detected. The intestines were normal. The stomach was found to contain brownish fluid, otherwise it was healthy. The liver was large, and inclined to be yellow. The kidneys were normal, with the exception of some small hæmorrhages in the cortex. The spleen was a little enlarged. There had been no hæmorrhages after the operation. There was no enlargement of the mesenteric glands. *The glands in the retroperitoneal and lumbar regions were enlarged and hæmorrhagic.* There were hæmorrhagic spots on the peritoneum which lay over them. The uterus was small, healthy, and normal. Ovaries and tubes normal. Pericardium contained some fluid, and there were hæmorrhages on its surface. The endocardium also contained hæmorrhages. The heart and its valves were strong and healthy, but the blood inside it was very dark and fluid. There was no clotting in the heart. Lungs were slightly congested at the bases, but otherwise normal. There were hæmorrhages on the pleura, but no pleuritic effusion. The brain was not examined. The gland in the groin was not hæmorrhagic. The smears from the gland were examined under the microscope, and were found to contain cocci. There was no satisfactory evidence of bi-polar organisms.

IX.—TREATMENT.

A. PROPHYLACTIC.

B. CURATIVE { *a. General.*
b. Specific. Serum Therapeutics.

A.—PROPHYLACTIC TREATMENT: Protective inoculation by the injection of Haffkine's prophylactic was confined almost entirely to the first outbreak in 1900. Apart from the two thousand inoculations performed at this time of panic, but few persons subsequently availed themselves of this measure of protection against attack or death. During more recent epidemics arrangements were made for placing Haffkine's vaccine at the disposal of health officers and others, but the indifference of the public towards inoculation led to its abandonment.

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* Note supplied by Dr. McLean, Medical Superintendent, Brisbane General Hospital.

Yersin's anti-plague serum was, however, frequently used in doses of 10 to 20 c.c., as a protective inoculation of the hospital, medical, and nursing staffs.

In the Maryborough outbreak the medical and nursing staffs were further protected by the wearing of specially devised overalls and respirators.

B.—CURATIVE TREATMENT:—

(*a*) *General and Medicinal:* The main indication for medicinal treatment is to counteract the effect of the virus in the system, and to maintain the strength of the patient. The symptomatic treatment of plague aims at the reduction of the temperature, the control of nervous excitement, the stimulation of the circulatory system, and the restoration of the powers during the stage of convalescence.

Cold and tepid spongings were often resorted to with excellent effects in cases with high temperature, and, as a rule, the patients reacted well to the sponging. The use of antipyretics for reducing temperature is. contra-indicated in plague; these drugs are frequently not only useless but dangerous. For insomnia and delirium, morphia given hypodermically in $\frac{1}{4}$ gr. doses was by far the best hypnotic, and was found to be safe and reliable. Trional and veronal were also found to be serviceable on occasions when combined with serum treatment.

As heart failure is the greatest danger of the disease, all severe cases were, as a matter of routine, treated with strychnine hypodermically administered.

Brandy, champagne, digitalis, strophanthus, carbonate of ammonia, atropine, caffeine, &c., were also found useful to increase the volume and tension of the pulse.

Good nursing and early confinement to bed of all cases are essential. The recumbent position was insisted upon until convalescence had been well established, and, as a rule, the patient was not allowed to sit up until the temperature had remained normal for several days.

The administration of antiseptics, such as carbolic acid, cyllin, permanganate of potash, &c., have been recommended with a view to counteracting the effects of the virus within the system. In our experience, the use of antiseptic drugs were found to exert no appreciable action, and they certainly did not possess the merit of the treatment by anti-toxic sera. With regard to the treatment of the bubo, it may be said that premature or too early surgical interference is to be deprecated. Upon the successful management of buboes depends to a large extent the period of detention of the patient in hospital. Premature incision of the bubo was found to lead to protracted, intractable, and infective sinuses, and to frequently necessitate the detention of a patient when otherwise convalescent. The bubo should be allowed to thoroughly mature before the abscess is opened, a clean cavity then being left which speedily granulates up. Suppuration also allows of the destruction of the specific bacilli by the pyogenic organisms.

Owing to the particular action of the plague virus upon the heart and circulation, sudden exertion on the part of the patient must be avoided, and on account of this tendency to fatal syncope, together with delirium and other nervous symptoms, plague of all diseases demands nursing by skilled and experienced attendants.

(*b*) *Serum Treatment:* The serum treatment of plague has been systematically carried out in Queensland since the beginning of the year 1901. The first consignment of Yersin-Roux serum, obtained from the Pasteur Institute of Paris, did not arrive until the end of July, 1900, and was, therefore, available for the latter part only of the outbreak of that year. In July, 1901, a further supply of Yersin's serum became available, and fresh supplies were regularly received each year up to 1904, when it was decided to obtain the serum from the Lister Institute of Preventive Medicine, in London. From 1904 to 1907 supplies have been received from the latter institute.

The immediate and remote results attending the administration of Yersin's serum have been, on the whole, of a satisfactory nature. The opinions expressed by various health officers, in charge of the patients at the Brisbane Plague Hospital from time to time, are to the effect that with serum given early, and in sufficient dosage, nearly every case of bubonic plague not attended with complications should get well. Our observations, covering a period of eight epidemic years, convince us that the early introduction of large doses of serum was attended with the best results. Its administration was quickly followed by a sudden fall of temperature, a moist skin, and profuse perspiration. There was also marked and rapid improvement in the mental condition, coma and even active delirium rapidly disappearing. The pulse and respiration were also favourably influenced by the use of the serum.

The routine treatment was to give an initial dose of 120 c.c. immediately the case was brought under observation. A second dose of 100 c.c. or more was given in twelve hours if the temperature still remained high; but it was rarely necessary to give a third dose. In the majority of cases the mode of administration was by subcutaneous injection, but where, from the gravity of the symptoms, severity of onset, or late stage of the illness, prompt measures were indicated intravenous injection was frequently resorted to. There was no selection of cases. As practically all cases treated at the Colmslie Plague Hospital, Brisbane, received serum treatment, strict statistical comparison cannot be made between the cases treated in the earlier stages of the disease and cases treated in the later stages. With regard to the latter cases, in which the prognosis was unfavourable, as also in patients exhibiting pulmonary or septicæmic symptoms, it is doubtful whether any considerable therapeutic effect was realised in the direction of diminishing the case-mortality.

The favourable impression we have been able to form in the use of any particular serum is not, however, based on the statistical results obtained in a series of plague cases treated with serum, as compared with an equal number of untreated and control cases. Probably the value of a serum can only be determined by such comparison.

Some indication, however, with regard to the efficacy of the sera used, can be arrived at by comparing the case-mortality of cases treated with serum and the case-mortality of cases not treated with serum in the records of the Plague Hospital cases published below. There was no reason to believe that the sera in any way aggravated the disease, or that any unfavourable influence had been exerted when serum was therapeutically administered to patients actually suffering from plague, or only presenting symptoms suggestive of plague. The conflicting opinions expressed by some clinicians as to the protective or curative properties exhibited by the various sera may find explanation in the mode of administration and the dosage recommended, or, what is, perhaps, more likely, in the method of preparation and varying potency of the sera employed. That fresh consignments of sera obtained by us from the institutes did vary in potency the series of experiments carried out by us on animals furnish statistical proof. The results of these experiments are given below. In connection with the question of the comparative efficacy of the various sera used by us and their variation in potency, the methods employed in the preparation of the sera, and the idiosyncrasy of the horses furnishing the sera, may possibly suggest an explanation of the varying results obtained. All sera furnished to us were carefully stored in cool chambers at about 40° F. There is a tendency of all sera to lose their specific properties when stored for any length of time, and the serum obtained from the Pasteur Institute in 1903 contained so large an amount of sediment that filtration through sterile wool warmed to body temperature had to be resorted to. The removal of the sediment did not appear to affect the potency of the serum, the clear filtrate when injected intravenously affording marked relief in many severe cases.

Of *local* effects produced by the injection of Yersin's serum fever, urticaria, erythema, œdema, local and general rashes, and joint pains supervened in a fair proportion of cases. Where the serum was used prophylactically in small doses, as in the case of nurses, doctors, and others exposed to infection from cases exhibiting pulmonary symptoms, rashes and joint pains often occurred in very pronounced form. The occurrence and severity of the rashes and joint pains seemed to be independent of the amount of serum administered. In the case of patients receiving large and repeated doses of the serum, local and general rashes with pains in the joints and muscles were frequently observed. The general rashes were urticarial, erythematous, scarlatiniform, and purpuric in character. In cases with marked urticaria the mucous membranes of the mouth, throat, eyes, and even the urethra were involved. In the treatment of the rashes calcium chloride in 20 gr. doses for three or four doses gave marked relief; while aspirin, phenacetin, and salicylate of soda appeared to alleviate the pains in the joints and muscles.

Mortality.—The results of the treatment of plague patients with sera in the Brisbane Plague Hospital for the epidemic years 1900-1907, inclusive, were as follow:—

1900.—Of 56 persons attacked, 25 died—a mortality of 44·6 per cent. No serum was then available.

1901.—Of 36 persons attacked, 12 died—a mortality of 33·3 per cent.
28 cases treated with serum—22 recovered, 6 died: 21·4 per cent. mortality.

1902.—Of 82 persons attacked, 26 died—a mortality of 31·7 per cent.
69 cases treated with serum—54 recovered, 15 died: 21·7 per cent. mortality.

1903.—Of 21 persons attacked, 11 died—a mortality of 52·3 per cent.
16 cases treated with serum—9 recovered, 7 died: 48·7 per cent. mortality.

1904.—Of 30 cases of plague, 8 died—a mortality of 26·6 per cent.
25 cases treated with serum—19 recovered, 6 died: 24·0 per cent. mortality.

1905.—Of 28 persons suffering from plague, 15 died—a mortality of 53·5 per cent.
21 cases treated with serum—11 recovered, 10 died: 47·6 per cent. mortality.

1906.—Of 11 persons attacked, 7 died—a mortality of 63·3 per cent.
7 cases treated with serum—3 recovered, 4 died: 57·0 per cent. mortality.

1907 (to 30th June).—Of 36 persons attacked, 14 died—a mortality of 39 per cent.
32 cases treated with serum—21 recovered, 11 died: 34·4 per cent. mortality.

The general results obtained are set forth in the following table:—

Year.	TOTAL CASES AND DEATHS.			SERUM-TREATED PATIENTS.			UNTREATED CASES.			Difference in Favour of the Serum-treated Patients.
	Number.	Deaths.	Case Mortality.	Number.	Deaths.	Case Mortality.	Number.	Deaths.	Case Mortality.	
			Per cent.			Per cent.			Per cent.	Per cent.
1900	56	25	44·6	No serum available			...	...	...	...
1901	36	12	33·3	28	6	21·4	8	6	75·0	53·6
1902	82	26	31·7	69	15	21·7	13	11	84·6	62·9
1903	21	11	52·3	16	7	43·7	5	4	80·0	36·3
1904	30	8	26·6	25	6	24·0	5	2	40·0	16·0
1905	28	15	53·5	21	10	47·6	7	5	71·4	23·8
1906	11	7	63·3	7	4	57·0	4	3	75·0	18·0
1907	36	14	39·0	32	11	34·4	4	3	75·0	40·6
TOTALS ...	300	118	39·3	198	59	29·7	46	34	73·9	44·2

The following tables set forth in tabular form—(1) An abstract of the cases of plague which were treated with serum; and (2) details regarding the serum injection, amounts of serum administered, the results obtained, and other particulars in each case:—

ABSTRACTS OF CASES TREATED WITH, AND WITHOUT, SERUM AT THE COLMSLIE PLAGUE HOSPITAL, BRISBANE, FOR YEARS 1900-1907.

1901.

No.	Sex.	Age.	Date of Admission to Plague Hospital.	How long Ill before Injection of Serum.	Total Quantity of Serum Injected. S. = Subcutaneously. I. = Intravenously.	Clinical Course.	Result.
1	M.	21	28 February	4 days	80 c.c.—S.	Right femoral bubo; rapid reaction to serum	Recovered.
2	F.	44	9 March	...	Nil	Right femoral bubo; albuminuria	,,
3	F.	23	11 ,,	4 days	100 c.c.—S.	Right femoral bubo	,,
4	M.	6½	15 ,,	...	Nil	Screaming delirium; *post mortem*, mesenteric glands hæmorrhagic	Died.
5	M.	...	17 ,,	5 days	100 c.c.—S.	Left inguinal bubo; delirium; suppuration of bubo	Recovered.
6	M.	29	23 ,,	2 ,,	180 c.c.—S.	Right femoral bubo; gradual onset; albuminuria	,,
7	F.	10	28 ,,	2 ,,	364 c.c.—S. + 40 c.c. I = 404 c.c.	Right femoral bubo; delirium; restlessness; high temperature; albuminuria; heart failure	Died.
8	M.	11	30 ,,	3 ,,	170 c.c.—S.	Left femoral bubo; sudden onset; delirium; marked reaction to serum; suppuration of gland	Recovered.
9	M.	21	6 April	2 ,,	160 c.c.—S.	Sudden onset; left femoral bubo	,,
10	F.	44	8 ,,	2 ,,	80 c.c.—S.	Sudden onset; left femoral bubo; suppuration of gland	,,
11	F.	42	8 ,,	3 ,,	200 c.c.—S.	Right femoral bubo; gradual onset; maniacal attacks	,,
12	M.	38	12 ,,	...	Nil	Bubo of left "groin"; hæmoptysis; heart failure	Died.
13	F.	49	19 ,,	3 days	100 c.c.—S.	Left femoral bubo; sudden onset	Recovered.
14	F.	25	20 ,,	7 ,,	340 c.c.—S.	Multiple buboes; gradual onset; delirium	,,
15	F.	...	28 ,,	2 ,,	320 c.c.—S.	Left femoral bubo; sudden onset; coma; suppuration of bubo	,,
16	M.	47	4 May	7 ,,	130 c.c.—S.	Left femoral bubo; gradual onset	,,
17	F.	25	9 ,,	5 ,,	500 c.c.—S.	Right inguinal bubo; pregnant, went full term; sudden onset; delirium; persistent vomiting; suppuration of bubo	,,
18	M.	2½	12 ,,	6 ,,	1060 c.c.—S.	Multiple buboes; sudden onset; delirium; meningitis; corneal ulcer	Died.
19	M.	26	9 ,,	5 ,,	200 c.c.—S.	Left inguinal bubo; delirium; cardiac depression	Recovered.
20	F.	4	11 ,,	3 ,,	80 c.c.—S.	Left pectoral bubo; sudden onset; suppuration	,,
21	M.	18	12 ,,	7 ,,	440 c.c.—S.	Right inguinal bubo; gradual onset; delirium; suppuration	,,
22	M.	21	18 ,,	..	Nil	Right femoral bubo; convalescent when admitted	,,
23	M.	16	17 ,,	...	Nil	Left femoral bubo; diagnosed *post mortem*	Died.
24	M.	17	20 ,,	2 days	820 c.c.—S.	Left femoral bubo; sudden onset; secondary buboes; coma; delirium; restlessness	,,
25	M.	25	28 ,,	4 ,,	100 c.c.—S.	Left femoral bubo; sudden onset; sudden collapse	,,
26	M.	26	29 ,,	4 ,,	430 c.c.—S.	Left femoral bubo; sudden onset; restlessness and delirium	,,
27	M.	32	13 ,,	2 ,,	400 c.c.—S.	Sudden onset; left inguinal bubo; delirium; suppuration of gland	Recovered.
28	M.	30	4 June	...	Nil	Septicæmic case	Died.
29	M.	24	25 ,,	...	Nil	Septicæmic case	,,
30	M.	19	28 ,,	4 days	720 c.c.—S.	Left femoral bubo; sudden onset; suppuration of bubo and development of secondary buboes; critical typhoid condition for two months	Recovered.
31	M.	27	3 July	1 day	380 c.c.—S. + 20 c.c. 6 days previous to attack	Bubo of right axilla; severe cardiac symptoms	,
32	M.	8	7 ,,	2 days	200 c.c.—S.	Left femoral bubo; sudden onset	Cured.
33	M.	37	24 ,,	3 ,,	200 c.c.—S.	Left femoral bubo; sudden onset; delirium	,,
34	M.	47	16 August	...	100 c.c.—S.	Left femoral bubo; unconscious	Died.
35	M.	33	2 October	...	Nil	Pneumonic case; collapse	,,
36	M.	19	9 December	7 days	140 c.c.—S.	Right femoral bubo; gradual onset; suppuration	Recovered.

Total number of cases	36	Total deaths	12	= 33·3	per cent.
Cases receiving serum	28	Deaths	6	= 21·43	per cent.
Cases untreated	8	Deaths	6	= 75	per cent.

1902.

No. of Case.	Sex.	Age.	Date of Admission to Plague Hospital.	How long Ill before Injection of Serum.	Total Quantity of Serum Used. S.=Subcutaneously. I.=Intravenously,	Clinical Course.	Result.
1	M.	14	27 January...	4 days	160 c-c.—S.	Left femoral bubo ; sudden onset : coma and delirium ; suppuration of gland	Recovered.
2	F.	47	...	..	Nil	Femoral bubo ; sudden onset ; sudden death ; diagnosed *post mortem* 3rd February	Died.
3	F.	48	...	...	Nil	Left femoral bubo ; diagnosed *post mortem* 7th February	,,
4	M.	33	..	..	Nil	Right inguinal bubo ; treated as typhoid fever ; diagnosed *post mortem* 9th February	,,
5	F.	39	8 February	2 days	180 c.c.—S.	No enlarged glands ; sloughing sore on right buttock ; plague organisms present ; pleurisy, pneumonia, and keratitis	Recovered.
6	M.	11½	17 ,,	2 ,,	100 c.c.—I. + 100 c.c —S.	Restlessness and delirium	,,
7	M.	27	...	...	Nil	Diagnosed *post mortem* 17th February	Died.
8	M.	15	19 ,,	2 days	300 c.c.—S. 190 c.c.—I. } = 490 c.c.	Left inguinal and femoral buboes	Recovered.
9	M.	11	15 ,,	7 ,,	160 c.c.—S.	Many glands affected ; sudden onset ; suppuration of submaxillary gland	,,
10	M.	19	20 ,,	2 ,,	240 c.c.—S. 210 c.c.—I. } = 450 c.c.	Left inguinal and femoral glands affected ; sudden onset ; suppuration of glands ; broncho-pneumonia ; albuminuria	,,
11	M.	15	22 ,,	1 ,,	180 c.c.—I.	Left inguinal bubo ; sudden onset ; delirium	Died.
12	M.	26	24 ,,	4 ,,	160 c.c.—S. 100 c.c.—I. } = 260 cc.	Left femoral bubo ; delirium ; broncho-pneumonia ; suppuration	Recovered.
13	M.	23	24 ,,	3 ,,	280 c.c. S. 110 c.c. I. } = 390 c.c.	Left femoral bubo ; sudden onset ; delirium ; albuminuria ; suppuration of bubo	,,
14	M.	14	26 ,,	2 ,,	100 c.c.—S. 80 c.c.—I. } = 180 c.c.	Right inguinal bubo ; sudden onset ; coma and delirium	,,
15	M.	6	26 ,,	5 ,,	280 c.c.—S. 240 c.c.—I. } = 520 c.c.	Many glands affected ; multiple abscesses ; gradual onset	,,
16	M.	14	5 March	3 ,,	200 c.c.—S. 240 c.c.—I. } = 440 c.c.	Left inguinal bubo ; sudden onset ; delirium ; suppuration of bubo	,,
17	M.	22	6 ,,	3 ,,	130 c.c.—I.	Left inguinal bubo ; sudden onset : suppuration of bubo ; albuminuria	,,
18	F.	33	8 ,,	2 ,,	220 c.c.—S.	Right femoral bubo ; sudden onset ; tarsal ulcer	,,
19	F.	20	10 ,,	5 ,,	120 c.c.—S.	Right femoral bubo ; sudden onset	,,
20	M.	20	9 ,,	1 ,,	120 c.c.—S. 370 c.c.—I. } = 490 c.c.	Right femoral bubo ; early heart failure	Died.
21	M.	46	10 ,,	8 ,,	180 c.c.—S.	Right femoral bubo ; delirium ; restlessness ; albuminuria ; suppuration of bubo	Recovered.
22	M.	38	10 ,,	(?)	120 c.c.—I.	Left femoral bubo ; died on day of admission	Died.
23	F.	40	10 ,,	2 days	100 c.c.—S.	Left femoral bubo ; sudden onset	Recovered.
24	M.	17	10 ,,	2 ,,	60 c.c.—S. 260 c.c.—I. } = 320 c.c.	Left femoral bubo ; sudden onset ; coma and delirium ; marked reaction to serum ; suppuration of bubo	,,
25	M.	9	10 ,,	1 ,,	320 c.c.—S. 160 c.c.—I. } = 480 c.c.	Left femoral bubo ; sudden onset ; meningitis	Died.
26	M.	17	11 ,,	2 ,,	200 c.c.—S. 140 c.c.—I. } = 340 c.c.	Right femoral bubo ; sudden onset ; delirium ; rapid reaction to serum ; suppuration of bubo	Recovered.
27	M.	41	12 ,,	3 ,,	120 c.c.—S. 260 c.c.—I. } = 380 c.c.	Left femoral bubo ; gradual onset ; suppuration of gland	,,
28	F.	20	19 ,,	4 ,,	100 c.c.—S.	Right femoral and inguinal buboes ; sudden onset	,,
29	M.	27	19 ,,	2 ,,	1780 c.c.—I.	Multiple buboes ; broncho-pneumonia ; delirium ; sudden onset ; suppuration of buboes	Died.
30	M.	19	19 ,,	2 ,,	240 c.c.—S.	Right femoral bubo ; sudden onset	Recovered.
31	M.	18	21 ,,	6 ,,	480 c.c.—S. 870 c.c.—I. } = 1350 c.c.	Multiple buboes ; sudden onset ; delirium ; broncho-pneumonia ; suppuration of buboes	Died.
32	F.	42	23 ,,	3 ,,	120 c.c.—S.	Right femoral and inguinal buboes ; sudden onset ; suppuration of primary bubo	Recovered.
33	F.	31	24 ,,	5 ,,	200 c.c.—S. 120 c.c.—I. } = 320 c.c.	Left femoral bubo ; sudden onset	,,
34	F.	16	...	...	Nil	Left femoral bubo ; diagnosed *post mortem* 30th March	Died.
35	F.	21	...	...	Nil	Left femoral bubo ; diagnosed *post mortem* 30th March	,,
36	M.	34	2 April	4 days	240 c.c.—S. 440 c.c.—I. } = 680 c.c.	Right inguinal buboes ; suppuration of bubo	Recovered.
37	F.	26	1 ,,	3 ,,	280 c.c.—S.	Left inguinal and femoral buboes ; miscarriage ; suppuration of buboes	,,
38	M.	40	8 ,,	4 ,,	100 c.c.—S. 200 c.c.—I. } = 300 c.c.	Right axillary bubo ; gradual onset	,,
39	M.	56	9 ,,	5 ,,	100 c.c.—S. 120 c.c.—I. } = 220 c.c.	Right and left inguinal buboes ; gradual onset	,,
40	M.	25	11 ,,	7 ,,	120 c.c.—S.	Multiple buboes ; sudden onset ; heart failure	Died.
41	F.	11	11 ,,	...	Nil	Unconscious ; diagnosed *post mortem* 11th April	,,
42	M.	28	13 ,,	2 days	120 c.c.—S. 280 c.c.—I. } = 400 c.c.	Left femoral bubo ; sudden onset ; delirium	Recovered.
43	M.	24	13 ,,	2 ,,	320 c.c.—S.	Right femoral bubo ; sudden onset	,,
44	M.	20	14 ,,	2 ,,	200 c.c.—S.	Right inguinal and femoral buboes ; sudden onset ; delirium ; suppuration of bubo	,,

1902—*continued.*

No. of Case.	Sex.	Age.	Date of Admission to Plague Hospital.	How long Ill before Injection of Serum.	Total Quantity of Serum Used. S. = Subcutaneously, I. = Intravenously.	Clinical Course.	Result.
45	M.	16	15 April	4 days	100 c.c.—S. 120 c.c.—I. = 220 c.c.	Right femoral bubo; sudden onset; suppuration of gland	Recovered.
46	M.	45	19 ,,	8 ,,	240 c.c.—S. 80 c.c.—I. = 320 c.c.	Left inguinal bubo; sudden onset; suppuration of bubo	,,
47	M.	17	18 ,,	2 ,,	220 c.c.—S. 120 c.c.—I. = 340 c.c.	Right femoral bubo; sudden onset; delirium; suppuration of bubo	,,
48	M.	60	19 ,,	4 ,,	220 c.c.—S. 120 c.c.—I. = 340 c.c.	Right femoral bubo; sudden onset; delirium; suppuration of bubo	,,
49	M.	14	21 ,,	3 ,,	160 c.c.—I.	Left femoral bubo; sudden onset; delirium; marked reaction to serum; suppuration	,,
50	M.	60	...	7 ,,	160 c.c.—S.	Pneumonic case; seen only a few hours before death on 22nd April	Died.
51	M.	32	23 ,,	2 ,,	120 c.c.—I.	Left inguinal and right femoral buboes; sudden onset; rapid reaction to serum	Recovered.
52	M.	16	24 ,,	1 ,,	60 c.c.—S.	Sudden onset; rapid reaction to serum	,,
53	M.	16	25 ,,	2 ,,	400 c.c.—S. 500 c.c.—I. = 900 c.c.	Right femoral bubo; sudden onset; coma; restlessness; delirium	,,
54	F.	50	...	?	Nil	Bubo; diagnosed *post mortem* 25th April	Died.
55	M.	15	25 ,,	4 days	240 c.c.—S.	Right femoral bubo; sudden onset; delirium; rapid reaction to serum; suppuration	Recovered.
56	F.	40	26 ,,	3 ,,	120 c.c.—S.	Right femoral bubo; sudden onset; suppuration of bubo	,,
57	M.	14	27 ,,	...	Nil	Left axillary bubo; very mild case	,,
58	M.	11 months	27 ,,	?	100 c.c.—S.	Right inguinal bubo; in dying condition on admission	Died.
59	M.	30	27 ,,	5 days	240 c.c.—S.	Right femoral bubo; sudden onset	Recovered.
60	M.	13	27 ,,	3 ,,	100 c.c.—S. 360 c.c.—I. = 460 c.c.	Right inguinal bubo; sudden onset; delirium	,,
61	M.	47	28 ,,	4 ,,	200 c.c.—S. 340 c.c.—I. = 540 c.c.	Right inguinal bubo; sudden onset; delirium; cardiac depression; typhoid conditions for two months	Died.
62	F.	14	29 ,,	2 ,,	160 c.c.—S. 120 c.c.—I. = 280 c.c.	Left femoral bubo; sudden onset; coma; delirium	Recovered.
63	F.	45	29 ,,	2 ,,	740 c.c.—I.	Pneumonic case, with bubo of left side of neck; sudden onset	Died.
64	M.	27	1 May	3 ,,	240 c.c.—S. 640 c.c.—I. = 880 c.c.	Left cervical bubo; pneumonic complication; sudden onset	,,
65	M.	55	5 ,,	6 ,,	420 c.c.—I.	Pneumonic case; gradual onset	,,
66	F.	13	...	...	Nil	Bubonic type; diagnosed *post mortem* 14th May	,,
67	M.	20	4 ,,	3 days	380 c.c.—S. 100 c.c.—I. = 480 c.c.	Right inguinal bubo; sudden onset	Recovered.
68	F.	45	5 ,,	4 ,,	120 c.c.—I.	Left axillary bubo; sudden onset	Died.
69	F.	6	6 ,,	2 ,,	320 c.c.—S.	Right inguinal bubo; sudden onset; delirium	Recovered.
70	F.	14	7 ,,	7 ,,	440 c.c.—S. 460 c.c.—I. = 900 c.c.	Right femoral bubo; double broncho-pneumonia; gradual onset	,,
71	M.	38	8 ,,	4 ,,	180 c.c.—I.	Right femoral bubo; sudden onset	,,
72	M.	26	9 ,,	...	Nil	Left femoral bubo; sudden onset; very mild course	,,
73	M.	40	9 ,,	4 days	160 c.c.—S.	Left femoral bubo; gradual onset; suppuration of bubo	,,
74	M.	27	11 ,,	1 ,,	240 c.c.—S. 160 c.c.—I. = 300 c.c.	Left parotid, and both submaxillary glands affected; sudden onset; delirium	,,
75	F.	52	16 ,,	7 ,,	320 c.c.—S. 120 c.c.—I. = 440 c.c.	Left femoral bubo; sudden onset; delirium; suppuration of bubo	,,
76	M.	43	15 ,,	...	Nil	Right inguinal bubo; sudden onset; admitted in dying condition	Died.
77	M.	32	16 ,,	6 days	280 c.c.—S. 360 c.c.—I. = 640 c.c.	Right femoral bubo; sudden onset; delirium	Recovered.
78	M.	38	26 ,,	5 ,,	460 c.c.—I.	Left femoral bubo; sudden onset; heart failure	Died.
79	M.	72	27 ,,	22 ,, ?	60 c.c.—S.	Left inguinal bubo; gradual onset; extensive valvular heart disease	Recovered.
80	M.	26	30 ,,	3 ,,	160 c.c.—S. 120 c.c.—I. = 280 c.c.	Left inguinal bubo; sudden onset	,,
81	F.	22	31 ,,	7 ,,	160 c.c.—S.	Right and left inguinal buboes; sudden onset; suppuration of buboes	,,
82	F.	30	4 August	...	Nil	Died on 5th August, at the General Hospital	Died.

Total number of cases	82	Total deaths	26 = 31·7 per cent.
Cases receiving serum	69	Deaths	15 = 21·74 per cent.
Cases untreated	13	Deaths	11 = 84·6 per cent.

1903.

No. of Case.	Sex.	Age.	Occupation.	Date of Admission to Plague Hospital.	How long Ill before Injection of Serum.	Total Quantity of Serum Injected C.C. S. = Subcutaneously. I. = Intravenously.	Clinical Course.	Result.
1	M.	26	Clerk	9 February, 1903	2 days	80 c.c.—S., 3rd day 80 c.c.—S., 4th day 100 c.c.—S., 5th day 120 c.c.—S., 6th day 80 c.c.—S., 3rd day	Temperature under 100° Fahr. 8th day Temperature normal 10th day	Recovered; discharged 21st March, 1903.
2	M.	22	Engineer	12 February, 1903	2 days	100 c.c.—S., 3rd day	Died 4th day	Generalised Pestis.
3	M.	17	Grocer's assistant	15 February, 1903	...	120 c.c.—S., 15 Feb., 1903 120 c.c.—S., 16 Feb., 1903 80 c.c.—S., 17 Feb., 1903 120 c.c.—S., 23 Feb., 1903 100 c.c.—S., 8 Mar., 1903 80 c.c.—7th day	Died 81st day	Plague septicæmia and exhaustion.
4	F.	8	Schoolgirl	19 February, 1903	7 days	100 c.c.—S., 8th day	Died 9th day	Generalised Pestis.
5	M.	31	Draper	Died at home	4 days	200 c.c.—S., 5th day	Died 7th day	Generalised Pestis.
6	M.	45	Produce merchant	11 March, 1903	4 days	120 c.c.—S., 5th day 120 c.c.—I., 8th day	Temperature under 100° Fahr. 11th day	Recovered; discharged 4th May, 1903.
7	F.	20	Printing office	12 March, 1903	2 days	320 c.c.—S., 3rd day 120 c.c.—S., 4th day	Temperature under 100° Fahr. 8th day	Recovered; discharged 28th April, 1903.
8	F.	11	Schoolgirl	14 March, 1903	1 day	160 c.c.—S., 2nd day 100 c.c.—S., 3rd day 120 c.c.—I., 3rd day	Temperature under 100° Fahr. 7th day	Recovered; discharged 2nd June, 1903.
9	F.	25	Domestic	23 March, 1903	5 days	120 c.c.—S., 6th day 120 c.c.—I., 7th day	Temperature under 100° Fahr. 8th day	Recovered; discharged 28th May, 1903.
10	M.	50	Employed at Bacteriological Institute	1 April, 1903	4 days	120 c.c.—S., 5th day 160 c.c.—I., 5th day	Temperature under 100° Fahr. 7th day	Recovered; discharged 10th May, 1903.
11	M.	12	Schoolboy	Children's Hospital, 5 April, 1903	2 days	Nil	Case recognised *post mortem* (diagnosed typhoid)	Death; Generalised Pestis; 5th day.
12	F.	29	Housewife	10 April, 1903	7 days	Nil	Temperature practically normal throughout	Recovered; discharged 13th June, 1903.
13	M.	30	Sanitary carter	20 April, 1903	4 days	120 c.c.—S., 5th day 120 c.c.—S., 6th day	Temperature under 100° Fahr. 8th day	Recovered; discharged 23rd May, 1903.
14	M.	20	City boot factory	24 April, 1903	2 days	200 c.c.—I., 3rd day 200 c.c.—S., 4th day	...	Death 9th day; Lobar pneumo-coccal pneumonia.
15	M.	19	City cement warehouse	26 April, 1903	9 days	200 c.c.—S., 10th day 120 c.c.—S., 11th day 160 c.c.—I., 12th day 160 c.c.—I., 13th day 200 c.c.—I., 16th day 120 c.c.—S., 20th day 140 c.c.—S., 21st day	...	Death 23rd day; Lobular plague broncho pneumonia.
16	M.	10	Schoolboy	2 May, 1903	2 days	200 c.c.—I., 3rd day 160 c.c.—S., 4th day	Temperature 100° Fahr. 3rd day	Death 5th day; cardiac failure.
17	M.	19	Carter	3 May, 1903	2 days	120 c.c.—S., 3rd day 160 c.c.—S., 4th day 200 c.c.—I., 5th day	Temperature under 100° Fahr. 8th day	Recovered; discharged 13th June, 1903.
18	F.	12	Schoolgirl	16 May, 1903	1 day	160 c.c.—S., 2nd day 160 c.c.—S., 3rd day	Temperature under 100° Fahr. 5th day	Recovered; discharged 13th June, 1903.
19	M.	33	Chinese fruiterer	General Hospital, 23 May, 1903	..	Nil	Case diagnosed *post mortem*	Death; Generalised Pestis.

Total number of cases ... ... *21 Total deaths ... ... 11 = 52·3 per cent.
Cases receiving serum .. ... 16 Deaths ... ... 7 = 48·7 per cent.
Untreated ... ... 5 Deaths ... ... 4 = 80·0 per cent.

*(2 cases diagnosed *post mortem.*)

1904.

No. of Case.	Sex.	Age.	Occupation.	Date of Admission to Hospital.	How long Ill before Injection of Serum.	Total Quantity of Serum Injected. S. = Subcutaneously. I. = Intravenously.	Situation of Buboes.	Clinical Course.	Remarks.
1	M.	55	Saddler ...	10 February	4 days ...	120 c.c.—S., 5th day	Right femoral region	Initial temperature, 103°; normal 4th day	Discharged 17th March, 1904.
2	M.	11	Schoolboy ...	11 February	2 days ...	80 c.c.—S., 3rd day 120 c.c.—S., 4th day	Left femoral region	Initial temperature, 103°; normal 10th day; pulse weak and dicrotic	Discharged 15th March, 1904; severe case.
3	F.	21	Bagmaker ..	17 February	3 days ...	120 c.c.—S., 4th day	Left femoral region	Temperature normal 18th day; albumen in urine	Discharged 19th March, 1904.
4	F.	7	Schoolgirl ...	18 February	1 day ...	120 c.c.—S., 2nd day 120 c.c.—I., 4th day	Left femoral region	Initial temperature, 105°; pulse weak and dicrotic; temperature normal 17th day	Discharged 19th March, 1904; very severe case; 120 c.c. intravenously.
5	M.	34	Bag and bale hawker	8 April ...	2 days ...	120 c.c.—S., 3rd day 120 c.c.—S., 4th day	Left femoral	Initial temperature, 104°; normal 5th day	Discharged 27th April, 1904.
6	M.	42	Produce hand	10 April ...	4 days ..	120 c.c.—S., 5th day 120 c.c.—S., 6th day	Left femoral region	Temperature normal 4th day	Discharged 27th April, 1904; 17 days only in Hospital.
7	M.	36	Produce hand	16 April ...	3 days ..	120 c.c.—S., 4th day 80 c.c.—S., 5th day	Left femoral region	Initial temperature, 104°; died on 2nd day in Hospital; complete renal suppression	Died 18th April, 1904; cultures of *B. pestis* were obtained from all the thoracic and abdominal viscera.
8	M.	32	Stable hand	Not removed to Hospital	1 day ...	200 c.c.—S., 2nd day	Left groin	Temperature normal 5th day	Recovered.
9	M.	16	Produce hand	27 April ...	2 days ...	120 c.c.—S., 3rd day 120 c.c.—S., 3rd day	Right groin, right femoral, and inguinal adenitis and periadenitis	Initial temperature, 99·2°; died on 2nd day after admission to Hospital	Died 28th April, 1904; general blood infection.
10	M.	16	Grocer and produce hand	3 May ...	3 days ...	120 c.c.—S., 4th day 200 c.c.—S., 5th day	Left femoral region	Initial temperature, 104·6°; temperature normal 20th day; gland sloughed *en masse*	Discharged 1st July, 1904; very severe case.
11	M.	28	Racehorse trainer	3 May ...	2 days ...	120 c.c.—S., 3rd day	Right femoral region	Initial temperature, 105°; normal 12th day	Discharged 8th June, 1904.
12	M.	22	Cook (oyster saloon)	5 May ...	4 days ...	120 c.c.—S., 5th day	Left femoral region	Initial temperature, 105·4°; pulse 152; dicrotic	Died on same day as seen, 5th May, 1904; general blood infection.
13	M.	25	Produce hand	9 May ...	2 days ...	120 c.c.—S., 3rd day	Right femoral region	Initial temperature, 102·6°; normal 6th day	Discharged 31st June, 1904.
14	M.	36	Produce hand	11 May ...	3 days ...	120 c.c.—S., 4th day	Left femoral region; gland sloughed *en masse*	Initial temperature, 104·2°; normal 9th day; there was a sore on the inner side of the left calf, which yielded plague bacilli on examination	Discharged 26th June, 1904; detained in Hospital 46 days; the longest case.
15	M.	34	Estate agent	15 May ...	6 days ...	120 c.c.—S., 7th day	Left femoral region	Temperature normal 7th day	Discharged 16th June 1904.
16	M.	19	Stableman ..	23 May ...	2 days ...	120 c.c.—S., 3rd day 120 c.c.—S., 4th day	Left femoral region	Temperature normal 5th day; serum rash	Discharged 14th June, 1904.
17	M.	8	Schoolboy ...	25 May ...	1 day ...	120 c.c.—S., 2nd day 120 c.c.—S., 3rd day	Right groin	Initial temperature, 105°; normal 6th day; pulse 136; weak and dicrotic	Discharged 26th June, 1904.
18	M.	14	Produce hand	25 May ...	5 days ..	120 c.c.—S., 6th day 120 c.c.—S., 8th day 120 c.c.—S., 9th day 120 c.c.—S., 10th day	Left inferior external axillary glands, also left and femoral gland	Initial temperature, 103·8°; delirium; cough and expectoration: respirations rapid; shallow for 7 days; albumen in urine; general rash; died 10th day	Died 5th June, 1904; developed intense blood infection and secondary specific pneumonia.
19	M.	17	Draper ...	31 May ...	4 days ...	120 c.c.—S., 5th day	Left femoral region	Temperature normal 9th day	Discharged 1st July, 1904.
20	M.	48	Baker ...	10 June .	2 days ...	120 c.c.—S., 3rd day 40 c.c.—S., 7th day	Left femoral region	Initial temperature, 104°; pulse 120; died 5 days after admission	Died 16th June, 1904; general hæmal infection; patient came from Ipswich.
21	M.	20	Student ...	12 June ...	3 days ...	120 c.c.—S., 4th day 120 c.c.—S., 4th day	Cervical glands, local lesion on nape of neck	Initial temperature, 104°; pulse 112; dicrotic; temperature normal 3rd day	Discharged 1st July, 1904; a remarkable case (*see* Appendix P).
22	F.	12	Schoolgirl ...	19 August ...	2 days ...	80 c.c.—S., 3rd day 120 c.c.—S., 4th day 100 c.c.—S., 6th day	Right femoral region	Temperature normal 9th day; delirium; weak, rapid pulse	Discharged 10th September, 1904.
23	F.	32	Housewife (mother of above case)	20 August ...	3 days ...	120 c.c.—S., 4th day	Right axillary, and slight swelling of left and right femoral glands	Temperature normal 7th day; severe headache	Discharged 10th September, 1904.
24	F.	39	Ragpicker ...	25 August ...	2 days ...	120 c.c.—S., 3rd day	Right inguinal	Severe headache and sleeplessness; weak, rapid dicrotic pulse	Discharged 10th September, 1904.
25	M.	19	Out of work	14 September	1 day ...	200 c.c.—S., 2nd day 160 c.c.—S., 3rd day	Right femoral region	Initial temperature, 104°, never lower than 100°; very delirious; diarrhœa; rapid, weak dicrotic pulse	Died 19th September, 1904.

Total number of cases	30	Total deaths	8 = 26·6 per cent.
Cases treated with serum	25	Deaths	6 = 24·0 per cent.
Cases untreated	5	Deaths	2 = 40·0 per cent.

1905.

No.	Sex.	Age.	Occupation.	Date of Admission to Hospital.	Time Ill before having Serum.	Total Serum.	Clinical Course.	Result.
1	M.	13	Schoolboy	26 February, 1905	1½ days	140 c.c.	Temperature reached 104·4 on 2nd day; normal 6th day. Albuminuria till 9th day. Noisy delirium for 3½ days	Discharged 26th Feb., 1905.
2	M.	17	Furniture storeman	27 January, 1905, from General Hospital	8½ days	240 c.c.	Temperature 104·4 9th day. Albuminuria 4 days after admission. Noisy delirium for 3 days after admission	Discharged 26th Feb., 1905.
3	M.	18	Plumber	30 January, 1905, from General Hospital	2 days ...	120 c.c.	Temperature 104·8 on 3rd day of illness. Died next day	Died 31st Jan., 1905.
4	M.	18	Grocer's hand ...	5 February, 1905	1 day ...	160 c.c.	Temperature 104·8 2nd day of illness; normal 6th day. No albumen	Discharged 9th March, 1905.
5	M.	59 ?69	Chemist's packer ..	5 February, 1905	3 days ...	160 c.c.	Temperature 104·4 4th day of illness; normal on 5th, but rose again. No albumen. Very intractable sinus after opening gland.	Discharged 25th March, 1905.
6	F.	25	Housewife	7 February, 1905, from General Hospital	1 day ...	240 c.c.	Temperature 105·4 on 2nd day; fell to normal next morning, but rose that night to same point, oscillating to 100·2 on 10th day; it reached 105·2 on 15th day. Mental condition clear to last	Died 22nd Feb., 1905.
7	M.	39	Dairy farmer ..	8 February, 1905	2 days ...	120 c.c.	Temperature 104; normal 9th day. No albumen	Discharged 8th March, 1905.
8	M.	28	Butcher	12 February, 1905	4 days ...	120 c.c.	Temperature 103·6; normal 10th day. Albumen disappeared on 8th day	Discharged 18th March, 1905.
9	M.	9	Schoolboy	14 February, 1905, from Children's Hospital	1 day ...	160 c.c.	Temperature rose to 105·2 on 4th day. Traces of albumen	Died 18th Feb., 1905.
10	F.	19	Home duties ...	16 February, 1905	Uncertain	240 c.c.	Temperature 104·4 on admission; fell nearly to normal on 4th day in hospital, but rose with increasing symptoms	Died 27th Feb., 1905.
11	M.	21	Storeman	16 February, 1905	3 days ...	100 c.c.	Temperature reached 103·8 on 4th day. Steady improvement, with trace of albumen on 10th day	Discharged 18th March, 1905.
12	F.	40	Home duties ...	16 February, 1905	...	Nil	Temperature 101·2 on admission. Gradual recovery	Discharged 25th March, 1905.
13	F.	49	Home duties ...	17 February, 1905	4 days ...	120 c.c.	Temperature 104·2; fell nearly to normal day after admission. No albumen. Dengue on 10th day	Discharged 11th March, 1905.
14	M.	12	Schoolboy	19 February, 1905	...	Nil	Temperature 102·2 after admission; normal second day. No albumen	Discharged 26th Feb., 1905.
15	M.	25	Saw-trimmer ...	28 February, 1905	2 days ...	240 c.c.	Onset with vomiting and headache. Perspired on 3rd day. Pulse, on admission, 104, and temperature 104·4, falling, after serum, nearly to normal midday, but rising immediately. Albumen. Many B.P. in blood before admission. Did not seem very ill till 2nd March, 1905	Died 2nd March, 1905.
16	M.	32	Fruiterer	6 March, 1905 (?)	2 days ..	240 c.c.	Pulse 110 on admission. Temperature 104·6, falling, after serum, nearly to normal, next day rising at once. Albumen. Very many B.P. in blood before admission. Did not seem very ill till shortly before death.	Died 7th March, 1905.
17	M.	19	Cowboy	9 March, 1905 (?)	8 days (?)	160 c.c.	Pulse 96. Temperature 102, falling to 101 after serum, but rising again to 102·8; normal on 3rd day in hospital. Much albumen	Discharged 25th March, 1905.
18	M.	25	Boot hand	4 April, 1905 ...	6 days ...	80 c.c.	Had rigor and sweating before admission. Pulse 100. Temperature 104·8 on admission, falling intermittently to normal on 7th day in hospital. No albumen.	Discharged 21st April, 1905.
19	M.	15	Produce store hand	Not admitted ...	1 day	20 c.c. 140 c.c. 160 c.c.	Delirium, pulse 120. Right femoral bubo. Died same day seen	Died 1st Jan., 1905.
20	M.	35	Storekeeper ...	,, ,, ...	...	Nil	No bubo. Septicæmic case. Was in dying state when seen by Health Officer at Brisbane General Hospital	Died 2nd Feb., 1905.
21	M.	20	Furniture warehouse employee	,, ,, ...	2 days ...	160 c.c.	Right axillary bubo. Extreme prostration. Pulse uncountable before removal to hospital	Died 2nd Feb., 1905.
22	F.	20	Domestic servant ...	,, ,, ...	...	Nil	A septicæmic case. Reported after death. *Post mortem* examination. All glands enlarged and hæmorrhagic, particularly the mesenteric	Died 7th Feb., 1905.
23	M.	30	Produce store hand	,, ,, ...	...	Nil	A septicæmic case	Died 24th Feb., 1905.
24	M.	14	Produce store hand	31 May, 1905 ..	1 day ...	120 c.c.	A septicæmic case	Died 2nd June, 1905.
25	M.	26	Grocer	Not admitted ...	...	Nil	A *post mortem* case	Died 15th May 1905.
26	F.	13	House duties ...	,, ,, ...	..	Nil	Died at home before seen	Died 16th May, 1905.
27	F.	49	Shopkeeper ...	15 June, 1905 ...	6 days ...	120 c.c.	Left upper femoral and inguinal buboes ...	Recovered.
28	F.	31	Household duties	15 June, 1905 ...	6 days ...	80 c.c.	Pulse 100 on admission, rising to 110 next day. Temperature 100 on admission, rising to 103 next day; normal on 10th day. No albumen	Died 1st July, 1905.

Total cases	28	Total deaths	15 = 53·5 per cent.
Cases receiving serum ...	21	Deaths	10 = 47·6 per cent.
Cases not treated	7	Deaths	5 = 71·4 per cent.

1906.

No. of Case.	Sex.	Age.	Occupation and Place of Employment.	Date of Report for Health Officer.	Date of Admission to Plague Hospital.	Length of time Ill before Injection of Serum.	Quantity of Serum used. S. = Subcutaneously. I. = Intravenously.	Clinical Course.	Result.
1	M.	25	Cellarman at a city warehouse	6 March ...	6 March	5 days ...	200 c.c.—S. ...	Very severe case. Great cyanosis at times, with difficulty in breathing, and blood-stained sputum. Repeated injections of strychnine and strophanthus necessary. Suppuration of bubo	Recovered. Discharged 25th April.
2	F.	27	Waitress in hotel, Stanley street, South Brisbane	26 March ...	31 March	4 days ..	100 c.c.—S. ...	Bubonic type. Temperature between 104° and 105° till 29th; sudden drop to below 100° on 30th. No subsequent rise. Much trouble with retention of urine	Recovered. Discharged from Private Hospital 28th April.
3	M.	33	Rag merchant, Lutwyche	13 April ...	14 April ...	3 days ...	80 c.c.—S. ...	Bubonic type of mild character. Ulcer on left buttock. Slight retention of urine. Suppuration of bubo	Recovered. Discharged 28th April.
4	M.	67	Fruit hawker, Fern street, Thompson Estate	13 April ...	...	...	Nil	Brought to Brisbane General Hospital in dying condition	Died.
5	M.	16	Butter packer ...	30 April ...	1 May ...	4 days ...	200 c.c.—S. ...	Very delirious; much vomiting	Died 3rd May.
6	M.	15	Working in slaughter-yards, Kedron	16 May ...	...	...	Nil	Reported *post mortem* ...	Died 16th May.
7	M.	15	Porter, Roma-street Railway Station	8 June ...	Not removed	...	Nil	Very mild case	Recovered.
8	M.	30	Cook, Hotel, Queen street	19 June ...	20 June ...	3 days ...	200 c.c.—S. ...	Delirious; passing urine and motions in bed	Died 21st June.
9	M.	22	Woodcutter, Leichhardt street, Spring Hill	18 November	Died before admission	1 day ...	120 c.c.—S. ...	Bubonic type, but appearance of blood-stained sputum, and rapid development of fatal symptoms	Died 19th November.
10	M.	50	Locker, H.M. Customs	23 November	23 November	7 days ...	120 c.c.—S. ...	Bubonic type. Apparently mild at first, but rapid development of fatal symptoms preceding death by only a few hours	Died 24th November.
11	M.	21	Hairdresser, George street, City	17 December	...	...	Nil	Reported *post mortem* ...	Died 17th December.

Total cases ... ... 11 Total deaths ... ... 7 = 63 per cent.
Patients receiving serum ... . 7 Deaths ... ... 4 = 57 per cent.
Untreated ... ... 4 Deaths ... ... 3 = 75 per cent.
(2 cases reported *post mortem.*)

1907.

No. of Case.	Sex.	Age.	Occupation and Place of Employment.	Date of Admission to Plague Hospital.	How long Ill before Injection of Serum.	Total quantity of Serum Injected in c.c. Subcutaneously.	Clinical Course.	Result.
1	M.	17	Clerk	3 January ...	1st day of illness	160 c.c.—S., 1st Jan., 1907, 1st day 160 c.c.—S., 2nd Jan., 1907, 2nd day 120 c.c.—S., 3rd Jan., 1907, 3rd day	Temperature normal in the a.m. 5th day, rose again; temperature normal 13th day	Cured; discharged 25th January, 1907
2	M.	39	Sailor	14 January ...	2 days ...	120 c.c.—S., 14th Jan., 1907, 3rd day	Temperature under 100° Fahr., 8th day. Ulceration of cornea	Recovered
3	M.	14	Schoolboy	16 January ...	2 days ...	120 c.c.—S., 16th Jan., 1907, 3rd day 120 c.c.—S., 17th Jan., 1907, 4th day	Died 5th day	Died 17th January, 1907, 4th day
4	M.	12	Schoolboy, and delivered milk	18 January ...	1st day of illness	120 c.c.—S., 17th Jan., 1907, 1st day 80 c.c.—S., 2nd day	Temperature under 100° Fahr., 3rd day	Cured; discharged 28th February, 1907
5	M.	10	Schoolboy	18 January ...	2 days ...	120 c.c.—S., 3rd day	Temperature under 100° Fahr., 7th day	Cured; discharged 28th February, 1907
6	M.	13	Schoolboy	18 January ..	1st day of illness	40 c.c.—S., 1st day 40 c.c.—S., 2nd day	Temperature under 100° Fahr., 4th day; normal 5th	Cured; discharged 22nd February, 1907
7	M.	10	Schoolboy	18 January ...	1st day ...	120 c.c.—S., 2nd day	Temperature under 100° Fahr., 3rd day	Discharged 22nd February, 1907; cured
8	M.	13	Schoolboy, and delivered milk	18 January ..	1st day ...	122 c.c.—S., 2nd day 80 c.c.—S., 4th day	Temperature under 100° Fahr., 7th day	Cured; discharged 2nd March, 1907
9	M.	14	Schoolboy, and assisting in grocer shop	18 January ..	3 days ...	120 c.c.—S., 4th day 80 c.c.—S., 6th day	Temperature under 100° Fahr., 15th day	Cured; discharged 2nd March, 1907

1907—*continued.*

No. of Case.	Sex.	Age.	Occupation and Place of Employment.	Date of Admission to Plague Hospital.	How long Ill before Injection of Serum.	Total quantity of Serum Injected in c.c. Subcutaneously.	Clinical Course.	Result.
10	M.	14	Schoolboy	20 January ...	6 days ...	120 c.c.—S., 7th day 80 c.c.—S., 9th day	Died 12th day	Died 25th January, 1907
11	M.	8	Schoolboy	20 January ...	1 day ...	120 c.c.—S., 2nd day 80 c.c.—S., 3rd day	Temperature under 100° Fahr., 8th day	Cured; discharged 2nd March, 1907
12	F.	14	Assists in housework	21 January ...	1 day ...	120 c.c.—S., 2nd day 80 c.c.—S., 4th day 80 c.c.—S., 5th day 80 c.c.—S., 6th day	Temperature under 100° Fahr., 9th day	Cured; discharged 2nd March, 1907
13	M.	40	Clerk, and assisting to clean Garnett's store	23 January ...	1 day ...	120 c.c.—S., 2nd day 80 c.c.—S., 4th day 120 c.c.—S., 8th day	Died 9th day	Died 30th January, 1907
14	F.	9	Schoolgirl	23 January ...	1 day ...	80 c.c.—S., 2nd day 80 c.c.—S., 3rd day 80 c.c.—S., 4th day	Temperature under 100° Fahr., 7th day	Cured; discharged 9th March, 1907
15	M.	49	Employed on rat-gang	26 January ...	1 day ...	120 c.c.—S., 2nd day 80 c.c.—S., 3rd day 80 c.c.—S., 3rd day 80 c.c.—S., 5th day 160 c.c.—S., 9th day 160 c.c.—S., 10th day	Temperature under 100° Fahr. on 7th day, and then rose again	Cured; discharged 22nd March, 1907. Re-admitted 25th March, 1907
16	M.	42	Employed on rat-gang	26 January ...	2 days ...	120 c.c.—S., 3rd day 120 c.c.—S., 7th day	Died 8th day	Died 31st January, 1907
17	M.	52	Employed on rat-gang	29 January ...	2 days ...	120 c.c.—S., 3rd day 80 c.c.—S., 4th day 80 c.c.—S., 6th day	Temperature under 100° Fahr., 8th day	Cured; discharged 5th March, 1907
18	M.	20	Driving baker's cart	1 February	1st day of illness	80 c.c.—S., 4th day 80 c.c.—S., 5th day 160 c.c.—S., 6th day 80 c.c.—S., 7th day	Died 10th day	Died 9th February, 1907
19	F.	57	Housework ...	2 February	3 days ...	120 c.c.—S., 1st day 160 c.c.—S., 4th day 80 c.c.—S., 6th day	Died 7th day	Died 5th February, 1907
20	M.	50	Carter	6 February	2 days ...	150 c.c.—S., 3rd day 80 c.c.—S., 5th day	Temperature under 100° Fahr., 6th day	Cured; discharged 5th March, 1907
21	M.	19	Packer, draper's warehouse	9 February	2 days ...	180 c.c.—S., 3rd day 80 c.c.—S., 4th day	Temperature under 100° Fahr., 6th day	Cured; discharged 5th March, 1907
22	M.	34	Station-owner ...	7 March ...	3 days ...	160 c.c.—S., 4th day 80 c.c.—S., 5th day 160 c.c.—S., 6th day	Temperature under 100° Fahr., 11th day	Recovered
23	M.	23	Collar-maker ...	12 March ...	2 days ...	160 c.c.—S., 3rd day 160 c.c.—S., 4th day 160 c.c.—S., 5th day	Temperature under 100° Fahr, 8th day; pulmonary complication	Recovered
24	M.	45	Cook, hotel ...	17 March ...	2 days ...	120 c.c.—S., 3rd day 80 c.c.—S., 5th day	Temperature under 100° Fahr., 7th day	Recovered
25	F.	14	Housewife, Church street, Valley	10 April ...	...	380 c.c.—S.	Left femoral bubo; delirium	Died
26	F.	45	Housework, Prospect street, Bowen Hills	9 April ..	...	160 c.c.—S.	Right femoral bubo; suppuration of bubo	Recovered
27	M.	7 d's	Mother died from pestis	13 April ...	3 days ...	4 c.c.—S. (prophylactic injection)	Septicæmic case	Died
28	M.	18	Cook, hotel in Queen street	25 May ...	2 days ...	240 c.c.—S.	Left inguinal bubo; suppuration of bubo	Recovered
29	M.	17	Yardboy, hotel in Queen street	28 May ...	2 days ...	320 c.c.—S.	Right femoral bubo; suppuration of bubo	Recovered
30	M.	8	Schoolboy, living at Hawthorne street, New Farm	Died at home	...	Nil	Left femoral bubo; diagnosed *post mortem*	Died
31	F.	75	Housework, Brown street, New Farm	Not removed	1 day ...	120 c.c.—S.	Left femoral bubo; sudden onset; too weak for removal	Died
32	F.	46	Cook and laundress, hotel in Creek street	Died in General Hospital	...	Nil	Septicæmic type; diagnosed *post mortem* 22nd February	Died
33	F.	23	Housework, Duncan street, Valley	Died in Lady Bowen Hospital	3 days ...	160 c.c.—S.	Left femoral bubo; confined of full term child; too ill to be removed	Died
34	M.	12	Flower-seller, Queen street	Died at his home in Wynnum	..	Nil	Right submaxillary gland; diagnosed *post mortem* 20th May	Died
35	M.	45	Publican, hotel, Queen street	...	..	Nil	Left femoral bubo; convalescent when seen	Recovered
36	M.	39	Telegraph operator, G.P.O., Brisbane	Died at home	1 day ...	120 c.c.—S.	Left femoral bubo; died suddenly before he could be removed	Died

Total number of cases to date, 25th June	36	Total deaths	14 = 39 per cent.
Cases receiving serum	32	Deaths	11 = 34·4 per cent.
Cases not treated	4	Deaths	3 = 75 per cent.

EXPERIMENTAL INVESTIGATIONS RELATING TO THE PROPERTIES OF ANTI-PLAGUE SERA.

All fresh supplies of sera obtained, and from time to time samples drawn from the stock in hand, were regularly tested.

A series of experiments were conducted by Mr. C. J. Pound, the Government Bacteriologist, with guinea-pigs, with a view of determining whether the particular sera in question exerted any effect in the direction either of curing, controlling, or protecting animals which received before, simultaneously, or subsequently with an inoculation of serum, an inoculation of virulent plague bacilli.

It will be seen by reference to the tables below that both Yersin's serum and the serum prepared at the Lister Institute, London, possessed in many instances remarkable curative and prophylactic properties.

The results of these experiments are incorporated in the following tables:—

TABLE A.—EXPERIMENTS TO TEST THE EFFICACY OF YERSIN'S SERUM ANTIPESTEUX, FROM A PROPHYLACTIC AND CURATIVE POINT OF VIEW; ALSO THE RELATIVE VALUE OF (1) THE SERUM AND ITS SEDIMENT, (2) CENTRIFUGALISED SERUM, AND (3) THE SEDIMENT FROM CENTRIFUGALISED SERUM.

1st day: "A1" guinea-pig injected with 10 c.c. serum and sediment.
2nd ,, 24 hours later inoculated with *B. pestis* from spleen of guinea-pig.
7th ,, Appears sick.
8th ,, Ruffled coat.
9th ,, Dull about the eyes; eating very little.
10th ,, Losing condition; gland suppurating.
12th ,, Slight improvement; eating better.
13th ,, Very much better.
16th ,, Still very thin, but eating well.
17th ,, Ruffled coat, and appears very sick.
18th ,, Died, 16 days after inoculation.

Inguinal glands suppurating, very few *B. pestis* present in the pus; spleen, liver, and kidneys very anæmic, and no *B. pestis* detected. Both lungs show large patches of necrosis, masses of *B. pestis*, very few bi-polar.

Serum culture from spleen and liver negative. Serum culture from lung, showing typical copious growth.

1st day: "A2" guinea-pig injected with 10 c.c. centrifugalised serum.
2nd ,, Inoculated with *B. pestis* from spleen of guinea-pig.
7th ,, Breathing quickly.
8th ,, Looks very sick; discharge in both eyes.
9th ,, Ruffled coat, and refused food.
11th ,, Still looking very ill; gland suppurating.
12th ,, Improving slightly.
13th ,, Very thin, but eating well.
16th ,, Appears quite lively.
17th ,, Decidedly improved; gland still suppurating.
18th ,, Still improving.
19th ,, Coat ruffled.
20th ,, Appears ill; breathing rapidly.
21st ,, Spasmodic breathing; very dull about the eyes.
22nd ,, Died, 20 days after inoculation.

Post mortem.—Small spleen, light-coloured, no necrosed patches; liver and kidneys anæmic; both lungs showed several large, hard necrotic areas. *B. pestis* numerous in lungs, few in pus from glands, but none detected either microscopically or by culture in spleen, liver, and kidney.

1st day: "A3" guinea-pig injected with sediment from 10 c.c. serum.
2nd ,, Inoculated with *B. pestis* from spleen of guinea-pig.
6th ,, Coat slightly ruffled.
7th ,, Eating well; does not appear sick.
8th ,, Looks well.
9th ,, Quite lively.
11th ,, Very lively and eating well, and in good condition.
12th ,, Still eating well.
16th ,, Losing condition; gland suppurating, few *B. pestis* detected; inoculations on culture (serum) media yield mixed growth, including *B. pestis*.
17th ,, Refusing food, getting very thin; gland very much enlarged; looking sick.
18th ,, Very uneasy; spasmodic breathing.
19th ,, Died, 17 days after inoculation.

Post mortem.—Few *B. pestis* in pus from enlarged gland. General anæmic condition of the muscles. Liver and spleen light colour, and atrophied. Lungs, several large areas of necrosis. *B. pestis* present in large numbers. No *B. pestis* detected in the spleen, liver, or kidneys.

1st day: "A4" guinea-pig (control).
2nd „ Inoculated with *B. pestis* from spleen of guinea-pig.
4th „ Ruffled coat.
5th „ Looking sick.
6th „ Very uneasy, and refusing food.
7th „ Eating very little; discharge in both eyes.
8th „ Died, six days after inoculation.

Post mortem.—Spleen very much enlarged and spotted. Liver friable and congested. Kidney and supra-renals congested. In both lungs several necrotic patches. Gland in the groin enlarged. *B. pestis* plentiful in all the organs and gland.

1st day: "B1" guinea-pig inoculated with *B. pestis* from spleen of guinea-pig, and simultaneously injected with 10 c.c. of serum and sediment.
5th „ Slightly ruffled coat.
7th „ Appears sick.
8th „ Looks very ill; refusing food.
9th „ Very thin, but appears brighter.
10th „ Very much brighter.
11th „ Very thin, but eating well.
12th „ Improving; gland suppurating, few *B. pestis* present in the pus.
13th „ Still very thin.
15th „ Improving, but still weak.
17th „ Very lively.
22nd „ Wound inside groin healed. Ultimately recovered.

1st day: "B2" guinea-pig injected with 10 c.c. centrifugalised serum, and simultaneously with *B. pestis* from spleen of guinea-pig.
4th „ Ruffled coat.
5th „ Breathing quickly.
6th „ Improving.
7th „ Eating very little food; losing condition.
8th „ Looking very sick.
9th „ Still very thin, but eating better.
10th „ Improving and getting lively.
11th „ Eating well.
15th „ Still very thin, but lively, and eating well.
17th „ Greatly improved.
18th „ Still very weak, but eating well.
19th „ Pus from gland contained a few *B. pestis*.
20th „ Wound healing.
21st „ Considerably improved; wound almost healed. Ultimately recovered.

1st day: "B3" guinea-pig injected with sediment from 10 c.c. serum, and afterwards inoculated with *B. pestis* from spleen of guinea-pig.
4th „ Ruffled coat.
5th „ Dull eyes.
6th „ Refuses food; very sick.
7th „ Gland suppurating; very ill.
8th „ Died, 7 days after inoculation.

Post mortem.—Spleen enlarged slightly, with few white spots. Liver congested. Lung shows few necrosed patches. Kidney and supra-renals slightly congested. Gland near site of inoculation ver much enlarged. *B. pestis* numerous in all organs and pus from gland.

1st day: "B4" guinea-pig (control) inoculated with *B. pestis* from spleen of guinea-pig.
3rd „ A little uneasy.
4th „ Ruffled coat; very dull-looking.
5th „ Discharge from both eyes; refusing food.
6th „ Spasmodic breathing.
7th „ Died, 6 days after inoculation.

Post mortem.—Typical plague appearance. Spleen large, necrotic. Liver dark colour and friable. Lung congested with few necrotic patches. Kidneys and supra-renals congested. Gland at seat of inoculation enlarged. *B. pestis* numerous in all organs and gland.

1st day: "C1" guinea-pig inoculated with *B. pestis* from spleen of guinea-pig that died of plague
2nd „ 24 hours later injected intraperitoneally with 10 c.c. of antipesteux serum and sediment.
6th „ Slightly ruffled coat.
7th „ Appears very uneasy.
8th „ Spasmodic breathing; discharge from eyes.
9th „ Very sick, and refuses food.
10th „ Died, 9 days after inoculation.

Post mortem.—*B. pestis* plentiful in pus at seat of inoculation. Lung, liver, and spleen contained numerous *B. pestis*.

1st day: "C2" guinea-pig inoculated with *B. pestis* from spleen of guinea-pig that died of plague.
2nd „ 24 hours later injected intraperitoneally with 10 c.c. of centrifugalised serum antipesteux.
5th „ Shows symptoms of illness.
6th „ Coat ruffled.
7th „ Takes very little food.
8th „ Breathing quickly.
9th „ Considerable discharge of pus from seat of inoculation.
10th „ Very ill, and refuses food.
11th „ Died, 10 days after inoculation.

Post mortem.—Pus from suppurating gland contained few *B. pestis*; spleen, lung, and liver contained *B. pestis* in large numbers.

1st day: "C3" guinea-pig inoculated with *B. pestis* from spleen of guinea-pig that had died of plague.
2nd ,, 24 hours later injected intraperitoneally with the sediment from 10 c.c. of antipesteux serum.
4th ,, Coat ruffled.
5th ,, Looking sick.
6th ,, Refusing food; still very ill.
7th ,, Pus from site of inoculation contained numerous *B. pestis.*
8th ,, Still looking very ill, but eating better.
9th ,, Slight improvement.
12th ,, Eating well, and appears more lively.
14th ,, Pus from suppurating gland contains few *B. pestis.*
16th ,, Considerable improvement.
20th ,, Pus from gland contained a few *B. pestis.*
24th ,, Pus from gland still contained a few *B. pestis.*
30th ,, Inoculation wound completely healed.
1st day: "C4" guinea-pig (control) inoculated with *B. pestis* from guinea-pig that had died of plague.
3rd ,, Coat ruffled; appears sick.
4th ,, Eyes almost closed, white discharge in the corners; very sick; refuses food.
5th ,, Died, 4 days after inoculation.

Post mortem.—Enlarged spotted spleen. Dark-coloured liver. Necrosis of both lungs. Congested supra-renals. *B. pestis* numerous in all internal organs and gland at seat of inoculation.

1st day: "D1" guinea-pig inoculated with *B. pestis* from spleen of guinea-pig that had died of plague.
3rd ,, 48 hours later, injected with 10 c.c. antipesteux serum and sediment.
5th ,, Ruffled coat; breathing rapidly.
6th ,, Refuses food; looking very sick.
7th ,, Died, 6 days after inoculation.

Post mortem.—*B. pestis* numerous in all internal organs and in the gland.

1st day: "D2" guinea-pig inoculated with *B. pestis.*
3rd ,, 48 hours later injected with 10 c.c. centrifugalised serum antipesteux.
5th ,, Looking sick; coat ruffled.
6th ,, Losing condition.
7th ,, Eats very little.
8th ,, Gland suppurating.
12th ,, Still very sick.
15th ,, Eating a little, but very thin.
16th ,, Still eating, very sleeply-looking, slight discharge around the eyes. Pus from suppurating gland contained *B. pestis.*
17th ,, Died, 16 days after inoculation.

Post mortem.—Spleen slightly enlarged and dark. Liver very much congested. Kidneys slightly congested. Lungs both slightly congested, left one having large hepatised area. Gland in groin breaking down with quantity of pus. *B. pestis* numerous, typical and involution forms in the lungs. No *B. pestis* in the spleen, liver, and kidneys. Few *B. pestis* and numerous cocci and other bacteria in the pus from gland.

1st day: "D3" guinea-pig, inoculated with *B. pestis* from spleen of guinea-pig that died of plague.
3rd ,, 48 hours later, injected intraperitoneally with sediment from 10 c.c. serum antipesteux.
4th ,, Appears sick; coat ruffled.
5th ,, Spasmodic breathing.
7th ,, Eats very little food: eyes very dull.
8th ,, Losing condition.
10th ,, Very sick.
11th ,, Very dull-looking, and refuses food.
14th ,, Still very sick and uneasy; gland suppurating and very painful.
16th ,, Dull about the eyes, slight discharge; quick spasmodic breathing.
18th ,, Considerable improvement: eating well.
19th ,, Pus from gland still contains few *B. pestis.*
20th ,, Very lively.
24th ,, Great improvement; increasing in weight. Gland still suppurating. Very few *B. pestis* present.
26th ,, Wound closing.
30th ,, Wound completely healed. From this time the guinea-pig rapidly recovered.
1st day: "D4" guinea-pig (control), inoculated with *B. pestis* from spleen of guinea-pig.
3rd ,, Dull look about the eyes.
4th ,, Refuses food; very ill.
5th ,, Died, 4 days after inoculation.

Post mortem.—Spleen teeming with *B. pestis.* Few *B. pestis* in liver, lungs and gland.

TABLE B.—FURTHER EXPERIMENTS WITH YERSIN'S SERUM.

"A3" guinea-pig injected with 2 c.c. Yersin's serum antipesteux (serum and sediment), and immediately after inoculated with *B. pestis* from spleen of guinea-pig.

"B3" guinea-pig (control) inoculated with *B. pestis* only.

Result.—"A3" remained apparently well, eating its food regularly until the 7th day; on the 8th day it showed signs of sickness; much worse on the 9th day, and died of plague on the 10th day.

"B3" (control) died of plague in 4½ days.

Four guinea-pigs, "A4," "B4," "C4," and "D4" (control), were each inoculated in the right groin with *B. pestis* from three-day-old serum culture from guinea-pig.

24 hours later "A4" was injected with 3 c.c. of Yersin's serum antipesteux (serum and sediment).

48 hours later "B4" was injected with ditto.

72 hours later "C4" was injected with ditto.

Result.—"A4" died in 11½ days after inoculation.

"B4" died in 11½ days after inoculation.

"C4" died in 6½ days after inoculation.

"D4" (control) died in 3½ days after inoculation.

In these experiments, although every animal inoculated with *B. pestis* died, the results are far from negative, for they show that in the unprotected guinea-pigs, death from plague took place in the usual time—viz., 3½ to 4½ days—while in those that received even the small dose of 3 c.c. of serum symptoms of illness were not manifest until the 6th day, and death did not take place until after the 10th day.

Briefly summarised, the results show that Yersin's serum antipesteux—even in a single dose—possesses remarkable protective and still greater curative properties. In those cases where, after receiving serum, the animals actually died of plague, death was very considerably delayed—in some instances until the 22nd day—while in unprotected animals death usually followed in from four to six days after inoculation.

From a prophylactic point of view, each of the guinea-pigs injected with Yersin's serum or sediment, and twenty-four hours afterwards inoculated with *B. pestis*, all died of plague, although the disease ran a very long course—in one instance the animal did not die until 22nd day after inoculation.

The animals inoculated with *B. pestis* and simultaneously injected with serum, or serum and sediment, recovered from an attack of plague after the 22nd day, while the guinea-pig simultaneously inoculated with *B. pestis* and the sediment of 10 c.c. of Yersin's serum died of plague on the 8th day.

The two most interesting and satisfying experiments are "C3" and "D3," where both guinea-pigs were inoculated with virulent plague bacilli, and subsequently—24 hours in one case and 48 hours in the other—injected with the sediment only of 10 c.c. of Yersin's serum antipesteux. Both animals became ill from the effects of the inoculation of plague bacilli, but both recovered completely.

The following series of experiments were made with samples of serum obtained from the Lister Institute of Preventive Medicine, London.

In each case when an animal died a *post-mortem* examination was held. A bacterioscopic examination of preparations from the blood, spleen, and glands, and inoculations on sterile serum media were also carried out. Unless otherwise stated, each experimental guinea-pig was an adult one, and, at the time of inoculation, well nourished and healthy, weighing approximately 850 grammes.

The inoculation was made with aseptic precautions subcutaneously inside the left thigh and the wound closed with surgical collodion; while the serum was injected intraperitoneally with a sterilised syringe. In each case a control animal was inoculated, the animals were examined daily, and their condition noted:—

TABLE C.—EXPERIMENTS MADE TO TEST THE PROTECTIVE AND CURATIVE POWERS OF LISTER INSTITUTE SERUM.

SERIES A.

Simultaneous Inoculation with B. pestis and Injection of Lister Institute Serum.

1. Two guinea-pigs inoculated with *B. pestis* and simultaneously injected with 10 c.c. serum.

Result: Each developed a suppurating bubo, became gradually sick, and, after continually refusing food for several days, died on the fourteenth and fifteenth day respectively after inoculation.

2. Two guinea-pigs were inoculated with *B. pestis* and simultaneously injected with 25 c.c. Lister serum.

Result: Both became very ill, developed enlarged glands, which suppurated. One died on the eighth and the other on the tenth day after inoculation. Control guinea-pig died of plague in four days.

SERIES B.

Serum used as a Protective.

1. Two guinea-pigs injected with 15 c.c. serum and twenty-four hours later inoculated with *B. pestis.*

Result: Both became very sick and emaciated, refusing food for several days, and developed suppurating buboes. One died of plague on the fourteenth day after inoculation, but the other, a much smaller animal, weighing 530 grammes, completely recovered.

2. Two guinea-pigs, each injected with 20 c.c. serum and twenty-four hours later inoculated with *B. pestis.*

Result: Both became ill with suppurating glands and very weak. One died on the twelfth day and the other on the twenty-third day after inoculation. *Post-mortem* examination showed *B. pestis* in each case. Control guinea-pig died of plague in five and a-half days.

SERIES C.

Serum used as a Curative.

1. Two guinea-pigs were each inoculated with *B. pestis* and twenty-four hours later injected with 15 c.c. serum.

Result: Both developed suppurating buboes, and became very sick and emaciated. One, weighing 470 grammes, died from exhaustion on the twenty-eighth day after inoculation. *B. pestis* were found in the pus from the gland, but no organisms were detected in the internal organs. The other guinea-pig, weighing 440 grammes, completely recovered.

2. Two guinea-pigs, each inoculated with *B. pestis* and twenty-four hours later injected with 20 c.c. serum.

Result: Both became very sick with enlarged glands. One died on the fifth day and the other on the seventh day after inoculation. *Post-mortem* examination revealed typical lesions and *B. pestis* in each case.

Serum as a Curative.

1. Two guinea-pigs were inoculated with *B. pestis*, and each animal was then injected with 40 c.c. of serum, half being administered on the first day and the remainder on the second day after inoculation.

Result: Both developed suppurating glands and became very thin and weak. One died on the sixteenth day, *post-mortem* examination revealing only a very few plague bacilli in the spleen and liver. The other, which weighed 450 grammes, gradually improved in condition and completely recovered. The control guinea-pig died of plague in four days.

2. One guinea-pig was inoculated with sputum from case of pneumonic plague, between the second and the fourth day, after 60 c.c. of serum was injected.

Result: Became very ill, refused food, developed suppurating gland, and died on the thirteenth day. The *post-mortem* examination revealed numerous *B. pestis* in the spleen.

SERIES D.

Experiments with Yersin Serum Antipesteux recently received from Gladstone.

(This serum was more than two years old.)

1. One guinea-pig injected with 20 c.c. serum and twenty-four hours later inoculated with *B. pestis.*

Result: Became very ill and developed suppurating gland, which almost healed. The animal became very emaciated, eating but little food, and died from exhaustion on the twenty-first day. The *post-mortem* examination revealed *B. pestis* in the pus, but no organisms were found in the internal organs.

2. One guinea-pig inoculated with *B. pestis* and twenty-four hours later injected with 20 c.c. serum.

Result: Developed enlarged gland, which commenced to suppurate on the sixth day, after which the animal refused food and became very thin and weak. Died on the twentieth day. *Post-mortem* examination revealed typical lesions of plague with *B. pestis* numerous in all internal organs. Control guinea-pigs for Series D died of plague in four days.

The conclusions arrived at from these experiments are as follows:—

Of fifteen guinea-pigs inoculated with virulent plague bacilli and injected before, simultaneously, or subsequently with varying quantities of Lister Institute Anti-plague Serum, twelve died of plague, while three, though obviously ill from that disease and losing fully one-fourth of their body weight, eventually completely recovered, regaining their normal weight and active condition within forty-five days of the date of inoculation.

Of the three recovered animals, two were inoculated with plague bacilli first, while the other one received serum first. Each of these three animals was little more than half the weight of those, with one exception, that died, which clearly demonstrates that the curative and protective value of anti-plague serum bears a very close relation to the body weight—*i.e.*, while a small animal of 450 grammes weight may be protected or cured with an injection of 15 c.c. of serum, a larger animal, weighing from 800 to 1,000 grammes, will require a correspondingly larger quantity of serum for protective or curative treatment.

The exceptional case referred to above was a guinea-pig weighing only 470 grammes, and, although it only received 15 c.c. of serum the day after it was inoculated with *B. pestis*, it survived until the twenty-eighth day; while the control, a large guinea-pig weighing 870 grammes, died of plague in five and a-half days.

One guinea-pig, which was inoculated with *B. pestis*, and, on the following day, injected with 20 c.c. of serum, died on the fifth day, the serum in this case having little or no effect whatever, as the animal must have been peculiarly susceptible. In the remaining cases which ended fatally the animals remained alive for varying periods, ranging from the second to the fourth week.

With reference to the experiments with Yersin's anti-plague serum, only 40 c.c. was available, therefore only two guinea-pigs were operated upon. The first received 20 c.c. serum, and on the following day was inoculated with *B. pestis.* It died from exhaustion, following a severe attack of plague, on the twenty-first day. The other was inoculated with *B. pestis*, then injected with 20 c.c. of serum on the following day. It died of plague on the twentieth day.

The results of these two experiments show that, notwithstanding its age—for it was considerably over two years old—this serum still possessed some immunising and curative properties.

The four control guinea-pigs all died of plague within five and a-half days.

X.—PROTECTION.

Mention has already been made of the measures undertaken for the prevention of epidemic plague. The scheme of action adopted may be briefly outlined as follows:—

1. Notification of all cases of plague, and of persons suspected to be suffering from plague.
2. Isolation of the sick.
3. Supervision of contacts.
4. Cleansing and disinfection of premises.
5. Destruction of rats.
6. Protective inoculation.
7. Fumigation of vessels.
8. Sanitary measures.

It will be seen that, in addition to the ordinary measures of isolation, segregation, disinfection, and sanitary improvement taken for the successful combating of epidemic diseases generally, it is necessary, in the case of plague, to include a special prophylaxis in view of the recognised and particular agents concerned in its spread.

First among measures required for the control of plague is prompt notification of every recognised case of plague, or any illness presenting symptoms suggestive of the disease. In order to ensure that plague in its more obscure manifestations should not be overlooked, notification of all cases of suspected plague was compulsory on the part of the medical practitioner attending the case. Directions for collecting and forwarding suspected material for bacteriological examination were issued by the Department to all the local authorities and their medical officers throughout the State. In the case of persons dying under circumstances pointing to plague as a probable cause of death, a *post-mortem* examination of the body was made, and material forwarded to the Government Bacteriologist for investigation.

International and interstate notification of plague, both in human beings and animals, and other details, were furnished regularly in the Official Bulletin issued each week by the office.

The removal to an isolation hospital of the sick was insisted upon, not so much on the ground of public safety (for the epidemic was not maintained either by direct or mediate communication of the sick to the healthy) but as a measure chiefly affecting the welfare of the patient himself, and those in close association with him.

The early removal to hospital not only offered the patient a better chance of recovery, so far as skilled nursing and constant medical attendance were concerned, but it also allowed of the invaded dwelling being adequately dealt with. As the incidence of the disease was on place and not on person, where one person contracted plague others could do so too. As plague is a disease common to both man and rats, the danger is not eliminated by the mere removal of the patient, if infected rats are still allowed to remain upon the infected premises. The partial evacuation of the dwelling, by the removal of the patient and other persons occupying the room or rooms, facilitates not only the cleaning and disinfecting operations necessary, but also permits a closer search of the patient's immediate surroundings for evidence of rat-infestation.

The segregation of contacts in bubonic cases of plague was early abandoned, and without any harmful results. By the term "contact" is meant a person who had been exposed to the same source of infection as the patient; not necessarily one who had only been in close association with the latter. In this respect, persons living in the same house with the patient may or may not be "contacts," if, as not unfrequently happened, the place of infection was proved to be the place of business and not the place of residence. Sometimes it was one, sometimes it was the other. In either case the danger attached to place and not to person. It was found impracticable to place restrictions on the movements of all contacts in the case of business establishments, hotels, public offices, &c. So far as the contacts of bubonic plague only were concerned, compulsory segregation was designedly abandoned. Where, however, patients exhibited symptoms of primary or secondary plague pneumonia, infection directly from person to person and possibly by indirect ways from infected human beings to rats was constantly borne in mind, and strict isolation of both patients and contacts carried out.

Immediately after the removal of the patient to hospital, disinfecting and cleansing operations were commenced. As a rule, not only the room lately occupied by the patient was disinfected but on occasion the whole house was operated on. A house-to-house search for the discovery of rats of the whole block or area was also undertaken.

In very few instances did infection recur subsequent to the disinfecting operations. This would appear to indicate that either the infection had been eliminated by the process adopted, or that it was merely of a temporary character.

Where premises were in such an insanitary condition as to render them incapable of effective disinfection, or so rat-infested as to constitute a danger, they were condemned as unfit for human habitation, and ordered to be pulled down. In a few cases they were destroyed by fire. An official report on the sanitary circumstances of the area was made by a trained and qualified sanitary inspector.

Efficient disinfection is a technical operation demanding some skill and special training on the part of the disinfector; and so far as the metropolis was concerned the disinfecting operations were directly supervised by a trained inspector, who also had the advantage of being a duly qualified chemist.

A variety of chemical disinfectants was used. In addition to the use of the ordinary solutions of perchloride of mercury, and carbolic acid or phenol, formalin sprays and vapour, and the burning of sulphur cakes were measures commonly adopted. Sulphuric and carbolic acids were used for soils and imperfect floorings; the earth beneath the floor being dug up to the depth of several inches. Fresh chlorinated lime in powder was largely strewn about in places likely to be frequented by rats, and also freely used for flushing drains, traps, gullies, &c. Of late, a number of comparatively new disinfectants have been placed on the market. Many of these latter preparations are claimed to be most powerful germicides, insecticides, and parasiticides, while at the same time they are said to be least poisonous to higher animal life. Experiments were made by the Government Bacteriologist to test the phenol co-efficiency—that is, the bactericidal power of a disinfectant in comparison with that of phenol or carbolic acid—of such disinfectants as Cyllin, Kreso, Izal, Kerol, Lysol, Cooper's fluid, &c., &c. With regard to the efficiency of these various disinfecting agencies to act as germicides when brought into contact with morbid secretions or excretions, some distinction would appear to be necessary between experiments carried out with pure cultures of "naked" bacteria grown artificially in the laboratory, and "natural" bacteria associated with organic matter existing in a natural medium.

So far as the disinfection of plague-infected premises is concerned, there can be little doubt that many of the present-day methods of house disinfection with perchloride of mercury, formalin, and sulphur are practically useless, and a waste of time, material, and money. In view of the fact that the rat-flea is a means by which plague is conveyed from rat to rat and rat to man, and that if a susceptible animal, such as the rat or the guinea-pig, be placed in a room with infected fleas the former may contract plague, it is manifest that chemical disinfection must be supplemented by agencies capable of acting both as germicides and parasiticides. In other words, the host or vehicle carrying the plague germ must be destroyed as well as the germ itself.

It is essential that spaces above the ceilings, between walls, and beneath floors be thoroughly searched for rats or their dead carcases, and that all cracks and crannies likely to harbour fleas come within the influence of the parasiticide used.

For reaching un-get-at-able places, and for spraying walls, fabrics, &c., we have found the "Tyree Automatic Spray" very effective. It is simple in construction, portable, and easy to work. The sprayer is worked automatically by a small charge of carbide of calcium which generates gas to eject the solution. For the fumigation of the holds of vessels nothing was found better than sulphur fumes. For the process to have a full measure of success it is necessary, however, to fumigate the hold before the cargo is put in. For the past six years an attempt has been made to prevent the access of shore rats to the ship, or ship rats to the shore, by a systematic fumigation of coastal vessels with sulphur before loading or after the discharge of cargo. Under interstate agreement all coastal vessels, coming from or touching at an infected port, are required to produce a certificate to the effect that fumigation has been carried out at the port of departure, and at the ultimate port touched on the voyage.

From 1st March, 1901, to 30th June, 1907, 228 oversea and 715 coastal vessels were fumigated at the port of Brisbane.

From 1st September to 30th June, 1907, 1,219 berthing-off certificates were also issued from Brisbane. "The Clayton Disinfecting Machine" has been much advocated for the fumigation of vessels, and from the limited experience we have had of testing the apparatus it would appear to have decided advantages over other methods. It consists of a combustion chamber for generating sulphur dioxide gas (dry), a cooling chamber, and a blower or fan to force the gas into the ship's hold. It combines efficiency with simplicity, and safety with economy. It is claimed that the machine disinfects and fumigates cargo without damage to the latter, and that it also exterminates all vermin and insect life aboard.

Of all the measures taken to control or prevent the spread of plague, the destruction of rats is the most essential, and, in many respects, the most difficult matter. The imperfections of the usual means hitherto employed, by trapping and baiting, have been fully recognised; and while statistical proof is forthcoming to show that large numbers of rats in a town have been destroyed and collected in any one year, it is still highly improbable that any one method of rat destruction has been attended with a marked diminution in the number of members of the rat tribe as a whole.

It may be said, however, that of all single prophylactic measures adopted for the suppression of plague the destruction of rats has been the one most uniformly successful. While it is extremely difficult, if not impracticable, to estimate the actual effect on the rat population produced by the procedure adopted, owing to the extraordinary fecundity of the rat and the difficulties attendant on the collection of all rats killed, it would be idle to suppose that the killing of rats and the investigation into the relation of dead infected rats have not been the most important of all factors in the so-called "stamping out" process of plague.

The dictum, "No Rats, No Plague!" would, indeed, supply a remedy as well as a warning, if the former could only be demonstrated on a scale approaching the method employed by the Pied Piper of Hamelin. Recognising the important part played by rats in the spread of plague, the Department has never relaxed the regular and systematic measures adopted both for the extermination of rats on shore and the precautions against their importation by ships. A trustworthy corps of twelve men (increased to twenty or fifty as circumstances demanded) under a competent inspector, was employed year after year, and every day of the year, whether plague prevailed or not, in Brisbane, in laying poisoned baits, trapping, digging out burrows, destroying nests, and breaking up the haunts of rats. Rat-catchers were also maintained, at the expense of the Government, in every port and important centre throughout the State all the year round.

Various chemical substances, such as phosphorus mixed with glucose, golden syrup, arrowroot, flour, &c., arsenic dissolved in alkaline broth, strychnine and arsenic pastes, &c., spread on small cubes of bread, or made up into little sandwiches flavoured with aniseed or oil of rhodium, were used for baits. The best results were obtained with the phosphorus bait. A smoke powder, consisting of arsenic 1 part, and sulphur sublimated 2 parts, used in an apparatus known as "The Universal Ant-killer," was especially effective when dealing with burrows or rubble-stone walls harbouring rats.

Attention has been given during the past few years to the destruction of rats by a virus, which latter is supposed to set up a virulent disease in the animals and quickly kill them. Several such bacteriological preparations are now on the market. The material consists of a nutrient medium (culture) containing the organisms. The preparation is said to have a fatal effect on rats, whilst being harmless to human beings and domestic animals. In order to test the value of the method the Department obtained supplies of the following preparations:—"Danysz" Rat Virus, "Laroche" Rat Virus, "Pasteur Vaccine Company's Virus," "Azoa," and "Ratin." In each instance the samples had lost in virulency, possibly due to a deterioration of the virus in course of transit from the laboratories abroad.

Only the "Danysz Virus" and the preparation "Azoa" were employed by us for rat-destruction to any extent. Experiments were commenced with a culture of the Danysz bacillus received from Paris on 4th May, 1905. From that date up to the present time the organisms have been kept up to a high state of virulence by passing them through the bodies of rats and mice by feeding and inoculation methods.

From 1st January to 30th June, 1907, the metropolitan area was systematically baited with the Danysz virus in conjunction with the phosphorus baits. The "Danysz" bait was always used on the day of preparation, and the area baited twice a week. Some 15,600 of these baits were laid during the above period; and of the rats collected in the ordinary way by the rat gang and forwarded for bacteriological examination 125 rats were found to have died from the effects of the Danysz virus. The result is not striking in view of the number of baits used and the extensive area operated on, but it is impossible to say what the actual results were in the open. Good results were reported from some of the Northern towns, where quantities of the virus had been supplied for treating large areas and the cane-fields. In many places a general exodus of rats was noticed from localities. Some seventy-six separate laboratory experiments were conducted with rats and mice (*see* Appendix A), the results of which experiments suggest that when the Danysz rat virus is constantly maintained at a high state of virulency the preparation possesses a great advantage over ordinary vermin killers. A properly prepared virus of a fresh and exalted nature should prove to be more effectual as a rat-destroyer in the future than it has appeared in the past. Every possible effort has been made by us to cope with the rat nuisance, and if the results have not always been commensurate with the expense and labour incurred by the Department it must, we think, be recognised that, considering the nature of the problem, of which no satisfactory solution has yet been found, to be dealt with, much has been done to lessen the danger from infected rats with the funds and staff available for the execution of this important measure of protection.

APPENDIX A.

REPORT BY THE GOVERNMENT BACTERIOLOGIST (MR. C. J. POUND) ON THE DESTRUCTION OF RATS BY A VIRUS.

(1.) REPORT ON SOME OBSERVATIONS BEARING ON THE EFFICACY OF THE DANYSZ' BACILLUS AS AN AGENT FOR DESTROYING RATS AND MICE.

Experiments were commenced with a culture of Danysz bacillus received from Paris on 4th May, 1905. From that date up to the present the organisms have been kept up to a high state of virulence by passing them through the bodies of rats and mice; in some instances by feeding, and, in others, by subcutaneous or intraperitoneal inoculation. Altogether, there were conducted 76 separate experiments, for which 39 mice and 98 rats were employed. Some of the animals were inoculated or fed with a pure culture of the bacillus, while others were treated with blood containing the bacillus from an animal recently dead of the disease.

The subcutaneous inoculations were carried out by making an incision in the groin, after sterilising the skin and introducing the infective material into the loose tissue by means of a platinum loop, while the intraperitoneal injection was affected by means of a hyperdermic syringe. In each case the wound was sealed by means of collodium.

In the majority of the feeding experiments young, virulent broth cultures were used. In some instances the broth was poured on to small cubes of bread, while in others the broth culture was thoroughly mixed with coarse oatmeal and then desiccated in a dry oven.

Feeding Experiments with Rats.—Of 60 rats fed with Danysz bacillus, 11 remained unaffected, while 49 died, in 45 of which the bacillus was detected and recovered by culture tests.

The shortest and longest periods in which death took place after feeding, and where the results were of a positive nature, were 2 days and 27 days respectively.

Of the four rats which died and no bacilli were detected, the deaths took place as follows:—One the second day, two on the third day, and one on the twenty-ninth day after feeding.

Inoculation Experiments with Rats.—Of 32 rats inoculated *subcutaneously* with Danysz bacillus 2 remained alive unaffected and 30 died, in 27 of which the characteristic bacillus and *post-mortem* appearances were present, and the bacillus recovered by culture tests.

The shortest and longest periods, when death took place with positive results, were 24 hours and 29 days respectively.

Of the three rats which died and no bacilli were found, one died on the second day, one on the eighth day, and one on the twentieth day.

Six rats were injected *intraperitoneally* with the bacillus. The shortest and longest periods in which fatality took place was 24 hours and 6 days respectively. *Post-mortem* appearances were typical, and the bacillus detected microscopically and recovered by culture tests.

Feeding Experiments with Mice.—Of 9 mice fed with the bacillus, 3 remained alive, while 6 died, with the usual *post-mortem* appearances. The bacillus was recovered by culture tests.

The shortest and longest periods in which death took place were 2 days and 12 days respectively.

Inoculation Experiments with Mice.—Of 29 inoculated *subcutaneously* all died with the usual *post-mortem* appearances. The bacillus was recovered by culture tests. The shortest and longest periods to the time of death were 24 hours and 13 days respectively.

Of two mice injected *intraperitoneally* with the bacillus, one died in 24 hours and the other in 5 days, both with pronounced *post-mortem* appearances. In each case the bacillus was recovered by culture tests.

For the cultures of the organisms a flask of strong broth is diluted with water, so as to charge four similar ones with 600 c.c. each. These are then sterilised in the autoclave, and, when cool, inoculated with a platinum needle loopful of a first or second generation agar culture; or, preferably, with spleen blood from an animal just dead of the disease.

The organisms, although they increase very slowly in the blood and tissues of the diseased rats, grow with marvellous rapidity in this artificial culture media.

After sixteen hours at 37 degrees C. the inoculated broth has a decided milky turbidity, when, by adding it to small pieces of bread, mixing it with pollard alone, or with dried mince meat, it is ready for use as baits. Recently, however, considerable success has been obtained experimentally by thoroughly mixing 600 c.c. of broth culture to 2 lb. of coarse oatmeal, and, after spreading it out in a thin layer, about ½-inch thick, on a large photographic dish, which is then placed in a drying oven or an incubator, kept at 35 degrees C. for about 12 hours, when the moisture will have evaporated, leaving the oatmeal plus the bacilli in a fairly desiccated state.

The bacilli in this dried condition retain their vitality and virulence with certainty for four weeks, and, in some instances, longer. Not only do rats and mice take this form of bait readily, but it has a decided advantage over the other forms of baits, inasmuch that it is not so quickly destroyed through contamination with adventitious bacteria and mould fungi as most baits would be.

Its distribution can be effected as with baits, by scattering the prepared oatmeal around the place inhabited by rats.

The results of the foregoing observations and experiments suggest that, when Danysz rat virus is properly prepared, and the virulence maintained, it should have a prominent place among the many methods which are at present being employed for rat destruction.

(II.) REPORT ON VIRUS "AZOA."

From these experiments it will be seen that mice are more susceptible to the effects of both inoculating and feeding with azoa.

Of three mice inoculated all died of the disease.

Of six mice fed with azoa, approximately six weeks old, all died of the disease.

Of four mice fed with azoa which was over two months old none died.

Of five rats inoculated three died of the disease.

Of eleven rats fed with azoa only one died of the disease.

In connection with these observations, attention must be directed to the fact that for several months considerable quantities of Danysz rat bacilli had been freely spread in and around Brisbane. Therefore, it is quite possible that some of the rats that are caught for the purposes of experiment may possibly have acquired immunity.

Experiments with Mice.

Two mice injected intraperitoneally. Result: Both died in 48 hours. *Post mortem* positive.

Mouse injected intraperitoneally with azoa over three months old. Result: Died in 24 hours. *Post mortem* positive.

Two mice fed on azoa. Result: One died in six days and the other in twelve days. *Post mortem* positive.

Two mice fed on azoa. Result: One died in eleven days and the other in twelve days. *Post mortem* positive.

Two mice fed with azoa. Result: Both died within ten days. *Post mortem* positive.

Two mice fed with azoa two months old. Result: Both remained alive.

Two mice fed on azoa three months old. Result: Both remained alive.

Experiments with Rats.

Two rats inoculated in the groin with azoa. Result: One died in eight days and the other in fifteen days. *Post mortem* positive.

Two rats injected intraperitoneally with azoa. Result: Remained alive.

Young rat inoculated. Result: Died in four days. *Post mortem* positive.

Rat fed on azoa. Result: Died in fourteen days. *Post mortem* positive.

Six rats fed on azoa remained alive. Ten days later refed. Result: Remained alive.

Two rats fed on azoa. Result: Remained alive.

One rat fed on a mouse recently dead of the disease. Result: Remained alive.

Rat fed on a mouse that had just died of the disease through inoculation. Result: Remained alive.

III.) A FURTHER REPORT ON THE RAT VIRUS "AZOA."

On the 3rd April last I received, per the Commissioner of Public Health, a sample of "Azoa," with instructions. Reports were made on the 23rd April and 16th July last.

During July I received a second sample with a further request for a report on same.

This rat virus was found to be composed of coarse oatmeal mixed with a broth culture of the bacillus typhi murium.

Inoculation Experiments with First Sample Received.

1. Two rats inoculated in the groin. One died in 8 days and the other in 15 days. *Post-mortem* positive.
2. One young rat inoculated subcutaneously. Died in 4 days. *Post-mortem* positive.
3. Five rats inoculated in the groin. Three died of the disease and 2 remained alive.
4. Two rats injected intraperitoneally. Both remained alive.
5. Two mice inoculated subcutaneously. Died in 24 and 48 hours respectively. *Post-mortem* positive.
6. Three mice inoculated at the root of tails. All died of the disease.
7. Two adult mice injected intraperitoneally. Both died of the disease within 48 hours.
8. Young mouse injected intraperitoneally. Died of disease in 24 hours.

Feeding Experiments—

9. Of 11 rats fed only 1 died of the disease.
10. Rat fed died of disease in 14 days.
11. Six rats fed. All remained alive. Refed 10 days later without producing any noticeable effect.
12. Two rats fed on 2 mice that had just died of the disease remained alive.

Inoculation Experiments with Second Sample, received July.

13. Five rats inoculated in the groin. Three died of the disease, while 2 remained alive.
14. Six rats injected intraperitoneally. Five died of the disease and 1 remained alive.
15. Twenty-five rats fed in 11 separate lots. Fourteen died of the disease and 11 remained unaffected.

In view of the fact that for over two years Danysz' Rat Virus has been spread systematically among the rat population of Brisbane, it is only reasonable to suppose that some of the rats, which were caught in various parts of the city, had recovered from the disease and were, therefore, immune to the "Azoa." However, these experiments demonstrate that the sample received in April was not up to the proper standard, while the July sample must be considered reliable and efficacious, as it killed 56 per cent. of the rats that were specially fed with it.

APPENDIX B.

Office of the Central Board of Health,
Brisbane, 28th January, 1901.

PLAGUE MEMORANDA.

TO ALL THE LOCAL AUTHORITIES IN THE STATE OF QUEENSLAND.

MEMORANDUM WITH RESPECT TO PLAGUE.

Sir,—In view of the possibility of cases of plague occurring again in Queensland, it is thought desirable to issue to the several Sanitary Authorities throughout the State of Queensland copies of memoranda, containing suggestions which may be of assistance to Local Authorities and their officers in guarding against the plague. Copies of these documents are enclosed.

Will you allow me to bring under your consideration the advantage which will result at the present juncture to the Sanitary Administration of having the active co-operation of the Local Authorities in the following directions:—

1. Whenever there is prevalence or threatening of plague, or any other epidemic disease, it is of more than common importance that the statutory powers conferred upon Local Authorities under "*The Health Act*, 1900," for the protection of the public health, should be well exercised by the Authorities acting with the advice of their Medical Officers of Health.

2. The system to be adopted for dealing with Plague should be thoroughly organised beforehand and the Authorities prepared for any re-invasion of the disease into their districts.

3. A continuation of measures for some period after the last case or cases have occurred is very essential to success in eradicating the diease.

4. It is always to be desired that the people should as far as possible know the real precautions they can take against the disease, what vigilance is needed with regard to its early symptoms, and what (if any) special arrangements have been made for giving medical assistance within the district.

5. Proper precautions are equally necessary for all classes of society, but it is chiefly with regard to the poorer population and dirtier quarters of the town that the Sanitary Authorities should exercise vigilance and tender information and advice.

6. The greatest safeguard is scrupulous cleanliness. The provisions of "*The Health Act*, 1900," dealing with Scavenging and Cleansing, section 65, should be discreetly but firmly enforced by the Local Authority and its officers. The scavenging of the district, the cleanliness of the surface of the ground, and the state of receptacles for excrement and garbage should receive active attention.

7. All conditions encouraging or facilitating the existence of rats, bugs, and fleas should be guarded against by every means within the power of the individual.

8. The powers which Local Authorities have to make By-laws with respect to lime-washing, cleansing, or purifying of uncleanly premises should be pressed with all practicable despatch.

9. Plague is a dirt disease, and the best defence of a town against the disease is abundant sunlight and fresh air. Where these are plentiful and cleanliness rules, experience shows that plague does not find a suitable soil for an epidemic spread.

10. In cases where the Local Authority has reason to suspect danger, house to house visitation by discreet and competent persons may be of the utmost service, both in quieting unreasonable alarm and in leading or assisting the less educated to do what is needful for safety; as well as in the discovery of unreported or suspicious cases of illness.

11. The extent of the coast line of Queensland, and the facilities for call at her numerous ports of oversea vessels, exposes the State to a more than usual risk of infection from the outside.

12. "It is to be remembered, however, that in proportion as a district is habitually well cared for by its Local Authority, the more formidable emergencies of epidemic disease are not likely to arise in it."

13. For the purpose of such information as is contained in this document and in the enclosed memoranda to Medical Officers of Health, printed handbills or placards may be usefully employed.

14. The measures suggested in the present memorandum are of an extemporaneous kind, and although Local Authorities should be on their guard against plague, it is not intended to suggest that there exists any cause for alarm at the present time.

I have the honour to be,

Your obedient servant,

B. BURNETT HAM, Commissioner of Public Health.

The Clerk to the Local Authority.

APPENDIX C.

Office of the Central Board of Health,
Brisbane, 28th January, 1901.

PLAGUE MEMORANDA.

TO ALL MEDICAL OFFICERS OF HEALTH OF THE LOCAL AUTHORITIES.

(I.)

Plague having for the space of 40 days receded from Queensland, a not unnatural, and to a great extent desirable, degree of apprehensiveness still exists regarding its reappearance in our State. The Medical Officers of the Local Authorities of Queensland will therefore need to be on the alert to detect the presence of this disease in their districts.

Though there does not exist any cause for alarm at the present juncture, the organisation for defence, however, requires to be placed on as complete and in as efficient a state of preparedness as is possible. In the event of plague being detected in any district, the measures to be taken to prevent its spread have been set out in the Regulations of the Central Board of Health, published in the *Government Gazette* of Saturday, 19th May, 1900. First among measures requisite for control of plague is prompt information to the Local Authority of all cases of the disease occurring in their district.

Under Section 134, sub-section 1 (*b*) of "*The Health Act*, 1900"—

(1) "Every medical practitioner attending on or called in to visit the patient shall forthwith, on becoming aware that the patient is affected with any infectious disease, or in the case of death from phthisis, within forty-eight hours thereafter, send to the Local Authority a certificate stating the name of the patient, the situation of the house, and the infectious disease with which, in the opinion of such medical practitioner, the patient is affected, or the fact of such death, as the case may be, and the Clerk of the Local Authority shall forthwith report the receipt of every such certificate to the Commissioner."

With a view to eliminate all conditions favourable to the existence or propagation of the disease indirect measures, such as the cleansing of dirty quarters of the town, are provided under Section 122, sub-sections 1, 2, 3, 4 of "*The Health Act*, 1900"—

(1) When a Local Authority is satisfied on the report of its medical officer of health, or of any medical practitioner, that the cleansing and disinfecting of any house or any part thereof, in the area, or of any articles therein likely to retain infection or contagion, would tend to prevent or check infectious disease, the Local Authority shall give notice to the owner or occupier of such house or part thereof, requiring him to cleanse and disinfect such house or part thereof and articles within the time specified in such notice to the satisfaction of the medical officer of health.

(2) If the person to whom notice is so given fails to comply therewith, he shall be liable to a daily penalty not exceeding ten shillings; and the Local Authority shall cause such house or part thereof and articles to be cleansed and disinfected, and may recover the expenses incurred by it in so doing from the owner or occupier in default.

(3) When the owner or occupier of any such house or part thereof is from poverty or otherwise unable, in the opinion of the Local Authority, effectually to carry out the requirements of this section, the Local Authority may cleanse or disinfect such house or part thereof or articles, and itself defray the expenses of so doing.

(4) The Local Authority shall enforce the provisions of this section in every case in which death from the disease known as phthisis is reported to it.

1. When plague threatens, the recognition of the first case is of paramount importance, for the history of epidemics teaches over and over again that a single case has contaminated a hitherto clean country. In times outside those of an epidemic, plague diagnosis is, more or less, a matter of probability, and the disease has not unfrequently been mistaken for typhus or typhoid fevers, pneumonia, or acute blood poisoning. Plague in these forms is always grave; not only because the "pneumonic" form is highly infectious to other persons, but for the reason that the milder varieties—*e.g.*, *Pestis minor*, or the *ambulant form* of the disease—by virtue of its mildness produces a false sense of security.

2. In order to help toward recognition of this disease in its obscurer manifestations, a statement of the clinical features exhibited will be found in Part II. of this Memorandum.

3. Cases of plague with such ill-defined clinical symptoms as referred to above are apt to elude diagnosis unless a bacteriological examination is additionally made on the patient or on the corpse.

With a view to assisting in the identification of plague newly developing in one or other district, the Central Board of Health has arranged for bacteriological testing, without cost to the Local Authority, of material submitted by their medical officer or medical officer of health from the earliest suspected case or cases.

Directions for obtaining and forwarding the suspected material will be found in Part III. of this Memorandum.

4. An essential measure of precaution in view of the observed relation between plague in *rats* and plague in the human subject, will be the prompt destruction of all rats in districts threatened or invaded by plague, care being taken that their carcases are collected and burnt without being unduly handled.

Any unusual sickness or undue fatality observed among rats should put the medical officer on his guard.

5. The isolation hospital should have a plentiful supply of fresh air and proper attention to cleanliness and disinfection, and when not occupied by any patients should be kept in good repair and the buildings well looked after by a competent caretaker.

6. The Central Board of Health having supplied themselves with a supply of Haffkine's Plague Prophylactic will be prepared to issue this material in limited amount on application of the Medical Officers of Health of districts actually invaded by plague.

B. BURNETT HAM, Commissioner of Public Health.

APPENDIX D.

Home Secretary's Department,
Brisbane, 1st November, 1906.

PLAGUE REGULATIONS.

HIS Excellency the Governor, with the advice of the Executive Council, has, in pursuance of the provisions of "*The Health Act of* 1900," been pleased to approve of the following Regulations made by the Commissioner of Public Health.

P. AIREY.

WHEREAS by "*The Health Act of* 1900" it is amongst other things enacted that in any emergency, of which the Commissioner of Public Health shall be the judge, the said Commissioner, in addition to all other powers vested in him, may, subject to the provisions of the said Act, make Regulations for house to house visitation, for medical aid and accommodation, for the inoculation of persons as a preventive against disease, for the examination, isolation, and accommodation of persons who are or are likely to be infected with endemic, epidemic, or infectious disease, for the speedy disposal of the dead, and for the promotion of cleansing, ventilation, and disinfection: And whereas an emergency has arisen requiring the making of Regulations with respect to the matters aforesaid by reason of the existence of certain infectious diseases in certain parts of Queensland and in other places from which such diseases may be introduced into Queensland unless proper precautions are immediately adopted: Now, therefore, the said Commissioner, with the approval of the Governor in Council, doth hereby make the following Regulations:—

PART I.

PRELIMINARY.

Title.

1. These Regulations may be cited as "*The Plague, Smallpox, and Cholera Regulations of* 1906."

Parts of Regulations.

2. The Regulations are divided into Parts, as follows:—

PART I.—PRELIMINARY;

PART II.—PLAGUE, SMALLPOX, AND CHOLERA;

PART III.—MISCELLANEOUS.

Duration, application, and execution of Regulations.

3. These Regulations shall continue in force until the first day of November, 1907, unless sooner rescinded or amended, and shall extend to and be in force in the Area of every Local Authority in Queensland.

The Health Officer shall superintend, and be charged with, the execution of these Regulations within his district.

Rescission of existing Regulations.

4. All Regulations heretofore made by the Commissioner of Public Health under the provisions of "*The Health Act of* 1900" for the treatment of persons affected with the infectious diseases known as plague, cholera, and smallpox, and for preventing the spread of any such disease, and all Orders of the Joint Boards for the Prevention of Epidemic or Infectious Diseases relating to such Regulations, are rescinded.

All offences committed against, and penalties incurred under, any of the said Regulations or Orders may be punished and recovered as if such rescission had not taken place.

Interpretation.

5. In these Regulations, unless the context otherwise indicates, the following terms have the meanings set against them respectively, that is to say:—

"Area"—Any City, Town, or Shire constituted under the laws in force for the time being relating to Local Government;

"Australasian State"—Any of the States of New South Wales, South Australia, Tasmania, Victoria, or Western Australia, or the Colony of New Zealand;

"Commissioner"—The Commissioner of Public Health appointed under the provisions of "*The Health Act of* 1900";

"Health Officer"—The Medical Practitioner appointed by the Governor in Council in any town or district for the purpose of "*The Health Act of* 1900";

"Local Authority"—The City, Town, or Shire Council, as the case may be, having jurisdiction within the Area;

"Master"—Any person, except a pilot, having, for the time being, command or charge of a vessel;

"Medical Officer of Health"—The Medical Officer of Health appointed by the Local Authority or other person performing his duties for the time being under the provisions of "*The Health Act of* 1900";

"Plague"—Bubonic or Oriental Plague;

"Cholera" includes Choleraic Diarrhœa;

"Infected Vessel"—A vessel on board of which there is a case of plague, smallpox, or cholera at the time of arrival at a port, or on board of which there has been a case of plague or cholera during the ten days immediately preceding such arrival; or a case of smallpox during the fifteen days immediately preceding such arrival;

"Suspected Vessel"—A vessel on board of which there has been a case of plague, smallpox, or cholera at the time of her departure from any port, whether in Queensland or elsewhere, or during the voyage, but on board of which there has been no case of plague or cholera during the ten days immediately preceding her arrival at a port, or a case of smallpox during the fifteen days immediately preceding such arrival;

"Ordinary Healthy Vessel"—A vessel not being an infected or suspected vessel, which has within the ten days immediately preceding her arrival at a port, left a place where plague or cholera is known to exist, or communicated otherwise than orally or by signal with any vessel which within that period has left such a place; or a vessel, not being an infected or suspected vessel, which has, within the fifteen days immediately preceding her arrival at a port, left a place where smallpox is known to exist, or communicated otherwise than orally or by signal with any vessel which within that period has left such a place;

"Healthy Vessel"—A vessel, even if arriving from a place where plague, smallpox, or cholera is known to exist, on board of which, whether before departure from such place or during the voyage, or on arrival at a port, no death from unknown or suspicious causes has occurred, and no case of such disease has occurred, and which has not within the ten days immediately preceding such arrival if arriving from a place where plague or cholera is known to exist, or within the fifteen days immediately preceding such arrival if arriving from a place where smallpox is known to exist—

- (*a*) Left a place where such disease is known to exist; or
- (*b*) Communicated otherwise than orally or by signal with any vessel which within that period has left a place where such disease is known to exist; or
- (*c*) Received any cargo from a place where such disease is known to exist.

And generally the terms used have the meanings respectively assigned to them by "*The Health Act of* 1900."

PART II.

PLAGUE, SMALLPOX, AND CHOLERA.

Cases of suspected plague, smallpox, or cholera to be reported.

6. When there is reason to suspect that any person is suffering from plague, smallpox, or cholera, the person in charge of the house or other premises where the person so suspected to be suffering then is, shall immediately report the fact to the Local Authority of the Area, who shall forthwith notify the health officer. If such person is on board of a vessel lying in a port, the master shall immediately report the fact to the health officer, or if he cannot make a report to such officer immediately, shall make a report immediately to a police magistrate or member of the police force, either of whom shall immediately report to the Commissioner.

When any case of plague, smallpox, or cholera, or suspected plague, smallpox, or cholera comes under the observation of a legally qualified medical practitioner, he shall immediately make a like report to the health officer, and also to the Commissioner.

Every Local Authority and health officer shall immediately report to the Commissioner every case of plague, smallpox, or cholera, or suspected plague, smallpox, or cholera reported to such Local Authority or health officer.

Premises or vessel to be isolated.

7. The Local Authority, health officer, police magistrate, or member of the police force, to whom any report is made as aforesaid, shall make immediate provision for preventing any person from leaving the premises where the person so suspected to be suffering then is, or where he resides, until the examination hereinafter prescribed has been made by the health officer, and for that purpose may station guards upon or near such premises, and use or cause to be used such force as may be necessary to prevent any person from leaving such premises.

The Local Authority, health officer, police magistrate, or member of the police force shall also prevent, by force if necessary, any person from entering such premises unless permitted so to do by the health officer.

The foregoing provisions apply as well to vessels as to premises on land.

Duty of health officer on report of case of plague, smallpox, or cholera.

8. The health officer, as soon as possible after the receipt of such report as aforesaid, shall examine the person who is believed or suspected to be suffering from plague, smallpox, or cholera, and also all other persons who are found upon the premises or vessel in which he then is, or who have recently been or are suspected to have been in contact with him during his illness.

The health officer, with respect to all or any of such other persons, may, for the purpose of observing whether they are suffering from plague, smallpox, or cholera, either—

- (i.) Order that they shall be detained for a period of five days or for such other period as the Commissioner may direct; a person so detained shall not be discharged until his person and personal effects have been disinfected to the satisfaction of the health officer; but if the health officer thinks fit, he may be discharged subject to the condition of inspection next hereinafter mentioned;
- (ii.) After disinfection of their personal effects discharge them upon condition that they report themselves to him and submit to inspection daily for a period of five days or for such other period as the Commissioner may direct.

Patients to be removed, where practicable, to hospital or other place appointed.

9. If the health officer certifies that any person on the premises or vessel is suffering from plague, smallpox, or cholera, he may be removed, if his condition will permit of such removal, to the isolation hospital or other place appointed for that purpose by the Minister. And no person so removed shall leave such hospital or place until the health officer has certified that he is free from such disease.

If any patient cannot be so removed, or for any reason it is not desirable to remove him, such premises or vessel shall remain subject, for the purpose of these Regulations, to the control of the health officer, and such person shall not be removed from or leave such premises or vessel, except with the permission in writing of the health officer.

Persons suspected of suffering from plague, smallpox, or cholera to be detained or removed.

10. If the health officer certifies that any person on any premises or vessel is suffering from an illness which the health officer suspects may prove to be plague, smallpox, or cholera, or that it is desirable that any such person should be detained for further observation, such person may either be detained in the premises or vessel where he then is, for such period as the health officer may direct, or may be removed to the isolation hospital or other place appointed for that purpose by the Minister, and detained there for a like period, in order that it may be ascertained whether he is or is not suffering from such disease.

No person so detained shall leave the premises, vessel, hospital, or place where he is so detained until the health officer has certified that he is free from such disease, or until his person and personal effects have been disinfected in manner hereinafter prescribed.

Health officer to order isolation and disinfection.

11. The health officer shall, under the direction of the Commissioner, in every case in which he gives a certificate under the two last preceding Regulations, give such directions and take such steps as may appear to him to be necessary for the purpose of isolating and disinfecting the premises or vessel where the person in respect of whom the certificate was given was found or where he resides. And all persons concerned shall obey any such directions.

The health officer may, under the direction of the Commissioner, cause any bedding, clothing, fittings, or furniture found on such premises or vessel to be disinfected in manner hereinafter prescribed, or to be destroyed.

He may in like manner cause any building or structure, which in his opinion is infected and which he certifies to to be incapable of proper disinfection, to be destroyed.

Health officer may examine premises or vessel without report made to him.

12. The health officer, if he has reason to suspect that any premises or vessel, whether reported to him or not, are or is infected with plague, smallpox, or cholera, may examine the same for the purpose of ascertaining whether such infection exists; and all persons concerned shall permit the same to be so examined, and shall obey all directions of the health officer in that behalf.

If the health officer, on making such examination, is of opinion that such premises or vessel are or is infected with such disease, the same consequences shall ensue as upon an examination made in pursuance of a report as hereinbefore provided.

Importation and transit.

13. Subject to the provisions of these Regulations, the following articles and goods liable to infection are prohibited from being brought into Queensland from any place where plague, smallpox, or cholera is known to exist, namely:—

(*a*) Body linen, wearing apparel, and other personal effects, and bedclothes which have been used.

But when such articles are in use or are carried as luggage or furniture they may be so brought into Queensland after being submitted to such special treatment as the Commissioner may direct.

(*b*) Rags and pieces of torn cloth, not excepting rags compressed by hydraulic power, which are carried as goods in bales.

(*c*) Used sacks, used carpets, used embroidery.

Provided that—

(i.) The transit of such articles or goods through a place where plague, smallpox, or cholera is known to exist, when packed in such a manner that they cannot be tampered with in transit, shall not prevent the same from being brought into Queensland.

(ii.) When such articles or goods have been carried in such a manner that they could not come into contact with infected or dirty articles in transit, their transit through a place where plague, smallpox, or cholera is known to exist shall not prevent the same from being brought into Queensland.

(iii.) This Regulation shall not apply to any such articles or goods if it is shown to the satisfaction of the Commissioner that they have been despatched five days at least preceding the first case of plague, smallpox, or cholera in such place.

No articles or goods shall be detained at the border line between Queensland and New South Wales.

Destruction of rats.

14. The health officer shall take all proper measures for the extermination of rats on board of vessels and on wharves and sea and river frontages and elsewhere within his Area.

The health officer shall indicate the area of operations of all rat-catching gangs employed by the Commissioner.

Disposal of rats taken.

15. The bodies of all rats caught or found dead shall in every case be forthwith destroyed by fire.

Precautions against plague in respect of vessels while in port.

16. It shall be the duty of every owner and master of every vessel in any port in Queensland to faithfully observe all the following provisions, namely:—

(i.) To keep the vessel off from the wharf or pier by a distance of at least four feet by means of fenders or rafts, and to prevent listing towards the wharf or pier.

(ii.) To place and keep placed on both ends of every hawser and rope connecting the vessel with the wharf or pier, a thick brush of metal bristles, or a funnel, or a stout metal disk at least twelve inches in diameter, and so constructed and so placed as to prevent migration of rats.

(iii.) To tar the fender and the fender-slings on such parts as to prevent migration of rats, and to keep the coal tar in a sticky condition by repeated applications. The coal tar should be mixed with a little oil or tallow.

(iv.) Any net used between the vessel and the wharf or pier to be removed between six o'clock in the evening and six o'clock in the morning, except only when cargo is being discharged or delivered during that time.

(v.) To draw entirely away and keep drawn entirely away all gangways when the same are not required for the discharging or the receiving of cargo from six o'clock in the evening to six o'clock in the morning.

(vi.) To suspend over the side along the wharf or pier four sets, or more if required, of electric or other lights, distributed so as to afford thorough illumination fore and aft along the whole length of the side of the vessel.

(vii.) To completely obstruct, by means of stout wire-netting, all pipes, ports, cabin scuttles, and other holes in the side of the vessel next to the wharf or pier, and to keep the same so obstructed while the vessel is alongside, in order to prevent both ingress and egress of rats.

(viii.) To prevent any refuse matter from being discharged into the port.

(ix.) Not to allow the landing of any rags or of any old cordage or second-hand clothing or bedding or of any used or returned produce bags whatever.

(x.) To thoroughly disinfect the bilges to the satisfaction of the health officer.

The Commissioner may, with the sanction of the Governor in Council, from to time suspend the operation of this Regulation for any period not exceeding three months, which period may, however, be renewed.

Disinfection, &c., of vessel on arrival.

17. All vessels arriving at a port from any infected port shall be subjected to the following treatment:—

(i.) Immediately after the discharge of cargo the master shall cause the vessel to be disinfected to the satisfaction of the health officer, when possible by means of steam sulphur fumes passed into the vessel under pressure, the sulphur being used in the proportion of one pound weight to every one thousand cubic feet of space, and the fumigation being continued for at least six hours, or by other equally effective means approved by the Commissioner.

(ii.) All such measures for the extermination of rats on board the ship as the Commissioner may direct shall also be immediately taken by the master.

Infected vessels.

18. All infected vessels arriving at a port shall be subjected to the following treatment:—

(i.) The sick shall be immediately landed and dealt with in manner hereinbefore described.

(ii.) The other persons shall be also landed, if possible, and detained or after disinfection discharged, subject to the condition of inspection hereinbefore prescribed. The period of such detention or inspection shall vary in accordance with the nature of the disease, the sanitary condition of the vessel at the time of arrival, and according to the date of the last case, but shall not exceed ten days in case of cholera and plague, and twenty-one days in case of smallpox.

(iii.) Soiled linen, personal effects in use, and articles belonging to the crew and passengers, which, in the opinion of the health officer, are contaminated, shall be fumigated, as hereinafter prescribed.

(iv.) The water in the hold shall, after such fumigation, be emptied.

(v.) Good drinking water shall be substituted for the water stored on board.

(vi.) All parts of the vessel which have been occupied by the sick or suspected shall be so fumigated.

(vii.) All cabins, and all parts of the vessel occupied by the sick or suspected, shall be emptied, and all articles disinfected in manner hereinafter prescribed.

The passages shall be disinfected by spraying or washing with solution of sublimate, hereinafter prescribed, with an addition of 10 per cent. of alcohol. The spraying shall be done by commencing with the upper part of the passage, following a horizontal direction, going down gradually in such a manner as to cover the whole surface with a layer of small drops.

The flooring shall be washed with the same solution.

Two hours thereafter the passages and the floor shall be scrubbed with water.

Suspected vessels.

19. All suspected vessels arriving at a port shall be subjected to the following treatment:—

(i.) All persons on board shall undergo medical inspection by the health officer.

(ii.) Soiled linen, personal effects in use, and articles belonging to the crew and passengers, which in the opinion of the health officer are contaminated, shall be fumigated as hereinafter prescribed.

(iii.) The water in the hold shall, after such fumigation, be emptied.

(iv.) Good drinking water shall be substituted for the water stored on board.

(v.) All parts of the vessel which have been occupied by the sick or suspected shall be similarly fumigated.

(vi.) The crew and passengers shall be prohibited from landing except by the permission, in writing, of the health officer, and then only upon condition of giving to him their names, their places of destination, and their addresses at those places.

(vii.) It shall be the duty of the master to cause all the provisions of this Regulation to be faithfully observed so far as the same are to be observed by him, his officers, or crew.

Pratique for vessels arriving from infected or suspected places.

20. No vessel arriving at a port from any place where plague, smallpox, or cholera is known or suspected to exist shall be admitted to pratique except between sunrise and sunset unless by special permission of the Commissioner.

Ordinary healthy vessels and healthy vessels.

21. Subject to the provisions of the last preceding Regulation, ordinary healthy vessels and healthy vessels shall be granted immediate pratique, whatever the nature of the bill of health may be.

Provided that the Commissioner may in the case of any ordinary healthy vessel direct that the treatment hereinbefore prescribed with respect to suspected vessels shall be observed.

And it shall be the duty of the master to cause all the provisions of this Regulation to be faithfully observed so far as the same are to be observed by him, his officers, or crew.

Filthy passengers.

22. When a vessel is not infected with plague, smallpox, or cholera, but has any passengers or crew on board who are in a filthy or otherwise unwholesome condition, the health officer may, if in his opinion it is desirable, with a view of checking the introduction or spread of such disease, give a certificate in duplicate in the following form or to the like effect, and shall deliver one to the master and transmit the other to the Commissioner :—

Certificate.

day of , 19 .

I hereby certify that I have examined the vessel from , now in the port of , and I find that she has passengers [*or* crew] on board in a filthy or otherwise unwholesome condition, and that in my opinion it is desirable, with a view of checking the introduction or spread of plague [*or* smallpox *or* cholera], that the persons on board the vessel should not be allowed to land unless they satisfy me as to their names, places of destination, and addresses at such places.

Health Officer.

When such certificate has been given no person on board the vessel shall leave or be allowed to leave the same unless or until he satisfies the health officer as to his name, place of destination, and address at such place, and such name and address shall forthwith be given by the health officer to the Commissioner.

Measures to be taken on the departure of a vessel from an infected place.

23. The following provisions shall apply to vessels on their departure from a place where plague, smallpox, or cholera is known to exist :—

(i.) The master shall not permit any person infected or suspected to be infected with any such disease to embark on the vessel. He shall not receive on board any bed-clothing, clothes, wearing apparel, or personal effects belonging to any such person.

(ii.) Before departure he shall cause the vessel to be put in a perfect state of cleanliness ; if necessary, he shall cause it to be disinfected in manner hereinbefore prescribed with respect to infected vessels.

(iii.) The drinking water taken on board shall be procured from a source well protected from all possible contamination. If the master is not satisfied that this is the case he shall cause all such drinking water to be distilled or boiled.

(iv.) If on arrival at a port the health officer is not satisfied that all the foregoing provisions have been complied with at the port of departure he may treat the vessel as an infected vessel.

Rules for carrying out fumigation operations.

24. The following rules shall be observed in carrying out fumigation operations :—

(i.) Wearing apparel, old rags suspected of being infected, papers, and other articles of no intrinsic value shall be destroyed by fire.

(ii.) Linen, bed-clothing, clothes, mattresses, carpets, and the like, infected or suspected so to be, shall be disinfected when possible by dry or moist heat.

Disinfecting solutions.

Disinfecting solutions may be :—

*(*a*) Solution of sublimate 1 to 1,000.

(*b*) Solution of pure crystallised carbolic acid or crude carbolic acid, impure, in a hot solution of soft soap, as 5 to 100.

(*c*) Milk of lime freshly prepared.

(*d*) Formalin, a 40 per cent. solution of the substance formaldehyde dissolved in water.

The following rules shall be observed in the use of disinfecting solutions :—

All persons who attend the sick shall wash their hands with the solution of sublimate or one of the carbolic solutions, diluted with an equal quantity of water.

The linen, clothes, and articles soiled by the excreta of the sick shall be dipped into the solution of sublimate. The 5 per cent. solution of pure carbolic acid or the solution of soapy carbolic may be used for the same purpose. The articles shall remain in the solution at least six hours.

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* (*a*) This solution will be coloured with aniline blue or indigo blue. It should not be put into metal dishes.

It is necessary to employ an acid solution of the sublimate, as a neutral solution is not destructive to microbes. Dissolve half-ounce of corrosive sublimate with 1 oz. hydrochloric acid (spirit of salts) in a bucketful (3 gallons) of hot water.

(*b*) Crude carbolic acid 5 pints, water 4 pints, caustic soda 1 lb. This makes about a 50 per cent. solution, and if added to ten times its bulk of hot water, or soft soap solution, makes a 5 in 100 (5 per cent.) solution.

(*c*) In order to have milk of lime in good condition, some quicklime of good quality should be taken and diluted by degrees with half of its weight of water. When the diluting is effected, the powder should be placed in a receptacle carefully corked up and in a dry place. To obtain a milk of lime which shall be nearly 20 per cent., it suffices to mix this in double its volume of water.

(*d*) Formalin is sold in bottles which hold 16 fluid ounces. For use, one part of formalin is to be mixed with nineteen parts of water—*i.e.*, one bottle of formalin and nineteen bottles of water, or one tablespoonful of formalin and nineteen tablespoonfuls of water.

All fabrics disinfected in the above solutions should be washed in clean water after disinfection.

All articles which will not stand, without deterioration, disinfection by heat—leather articles glued wood, felt, velvet, silk, and the like—shall be washed in the solution of sublimate. Coin may be disinfected with the soapy carbolic solution.

The carbolic solutions shall be used specially for disinfecting articles which can stand neither disinfection by heat nor the contact with sublimate, such as metals, instruments, and the like.

The milk of lime should be used for disinfecting the excreta and vomiting. Expectoration and purulent matter shall be destroyed by fire.

Disinfection.

25. Disinfection shall be compulsory for dirty linen, wearing apparel, clothing, and articles which form part of the luggage or furniture of any person coming from a place where plague, smallpox, or cholera is known to exist, and which the health officer may consider to be contaminated.

Fumigation shall only be applied to articles and goods which the medical officer of health considers to be contaminated, or to articles and goods the importation of which are prohibited under these Regulations except under the conditions hereinbefore mentioned.

The Commissioner shall fix the mode and place of fumigation.

The fumigation shall be carried out so as to damage the articles and goods as little as possible.

Letters and correspondence, printed books and newspapers, business papers, and the like, other than postal packages, shall not be liable to any restriction or fumigation.

Disposal of the dead.

26. It shall be the duty of the health officer to arrange for the speedy and efficient disposal of the dead.

In all cases of death from plague, smallpox, or cholera, the dead body shall, whenever it is practicable so to do, immediately after death be surrounded by a sheet soaked in a solution of sublimate of the strength hereinbefore prescribed, or carbolic acid, and placed in a coffin containing freshly burned and slaked lime, or other equally effective disinfectant approved by the Commissioner, and shall be surrounded therewith and shall be buried, when possible, in a deep and dry grave in a place which is not likely to be again used or disturbed, or, if buried in a cemetery, the coffin shall be surrounded by a layer of chlorinated lime at least 6 inches in thickness.

Isolation hospitals.

27. Where the isolation hospital is under the superintendence of the health officer, it shall be the duty of such health officer to undertake the care and treatment of the sick within such hospital, and generally to discharge such duties relating to the prevention of infectious disease as the Commissioner may direct. He shall also take all necessary steps for the promotion of the cleansing, ventilation, and disinfection of such hospital and all allied buildings, and he shall keep full records of the several cases which occur within such hospital.

Cleansing, &c.

28. The health officer shall supervise the work of all cleansing and disinfecting gangs.

PART III.

MISCELLANEOUS.

House to house visitation.

29. The health officer, with such sanitary inspectors as may be necessary, shall, at such intervals as occasion may require, make a house-to-house visitation within the limits of any locality from time to time defined by the Commissioner to be or to be likely to be subjected to a visitation of plague, cholera, or smallpox.

Medical examination.

30. Such health officer or other medical practitioner may medically examine all or any inmates of a house within such locality as aforesaid, and duly provide for the isolation and accommodation of persons who are likely to be infected with such disease.

Inoculation.

31. The Commissioner may make such arrangements as may be necessary to afford facilities for the inoculation of persons with Haffkine's prophylactic as a preventive against plague, or with freshly prepared calf lymph as a preventive against smallpox.

The health officer, the medical officer of health of the Local Authority, or other medical practitioner appointed by the Local Authority in that behalf, or the Commissioner, may inoculate any persons seeking inoculation as a preventive against infectious disease.

Bacteriological investigation.

32. Such measures may be taken, in accordance with the direction of the Minister, as may be necessary to ensure proper bacteriological investigation for determining the presence or absence of infection in any person, animal, or thing, and also to regulate the means of transport, without danger, of animals or things for the purpose of such investigation.

Regulations not to apply to vessels liable to quarantine laws.

33. Nothing in these Regulations shall be taken to apply to vessels coming from beyond sea and subject to laws in force for the time being relating to quarantine.

Penalty.

34. Any person who by any act or default commits a breach of any of the provisions of these Regulations shall be liable to a penalty not exceeding fifty pounds.

The foregoing Regulations were made by me on the nineteenth day of October, 1906.

B. BURNETT HAM, M.D.,
Commissioner of Public Health.

APPENDIX E.

DEPARTMENT OF PUBLIC HEALTH.

Year 190 .

Place..............................

PLAGUE.

Date .... Clinical form .... Case No. ....
Surname .... Christian Name ....
Age .... Sex ....
Residence ....
Employment, Place of ....
Nature of ....
Last Attendance at ....
Local Authority ....
Case Reported by Dr. .... Date Reported ....
Date of Visit of Health Officer ....
History of present Illness. Began on ....
at..........................m.; first noticed ....
Clinical features ....
....
....
....
Temperature .... Pulse .... Respirations ....
Bubo of ....region, first noticed on ....
as .... Size .... Tenderness ....
Consistence .... Periadenitis ....
Relation to Previous Cases ....
Relation to Infected Articles ....
Relation to Dead and Sick Rats ....
Relation to Fleas ....
Movements for Ten Days previous to Attack ....
....
....

RESULT OF CLINICAL EXAMINATION.

Positive .... Negative ....
Type of Disease ....
Any Pneumonic Symptoms ....
Date of Serum Injection { Subcutaneously ....
.... { Intravenously ....

BACTERIOLOGICAL INVESTIGATION.

Date Specimens obtained { Gland ....
{ Sputum ....
{ Blood ....
.... { *Post mortem* ....
Date Specimens examined by Health Officer ....
Positive .... Negative ....
Date Specimens examined by Government Bacteriologist ....
Positive .... Negative ....

OTHER INFORMATION.

Number of Contacts ....
Names and Addresses of Contacts ....
....
....
Date of Removal to Plague Hospital ....
Probable Source of Infection ....
Remarks ....
....
....
Date of Discharge or of Death ....

CERTIFICATE.

I, the undersigned, hereby certify that in my opinion ....
.... suffering from the Disease known as .... Plague.

Signature of Health Officer ....
Date ....

(To be filled in and forwarded to the Commissioner of Public Health, Brisbane.)

APPENDIX F.

TEXT OF POSTER ISSUED IN 1902.

OFFICE OF THE COMMISSIONER OF PUBLIC HEALTH.

NOTICE.

TO THE OCCUPIER OR OWNER OF THE PREMISES.

Sir or Madam,

Your serious attention is called to the following sections of "*The Health Act of* 1900":—

CERTAIN NUISANCES ON PREMISES.

65. Any person, whether the occupier or owner of the premises, who—

(1) Allows the CONTENTS OF ANY SANITARY CONVENIENCE, such as urinals, earth-closets, cesspits, ash-tubs, dust-boxes, &c., to overflow or soak therefrom; or

(2) Allows any WASTE WATER to run from any premises so as to cause an offensive smell; or

(3) Suffers any RUBBISH, FILTH, or UNWHOLESOME MATTER or thing to collect on any land abutting on a street and lying below the level thereof;

shall be liable to a PENALTY not exceeding FORTY SHILLINGS, and to a DAILY PENALTY not exceeding FIVE SHILLINGS.

The Local Authority shall cause every such nuisance to be abated, and may recover the expenses incurred by it in so doing from the occupier or owner of the premises on which the nuisance exists.

* * * * * * * * * * * *

REMOVAL OF FILTH.

67. When it appears to the Local Authority that any accumulation of manure, dung, soil, or FILTH, or other OFFENSIVE or NOXIOUS MATTER, ought to be removed, the Local Authority shall give notice to the person to whom the same belongs, or to the occupier of the land whereon it exists, or where there is no occupier, then to the owner of the land, to remove the same.

If such notice is not complied with within twenty-four hours from the service thereof, the manure, dung, soil, filth, or matter referred to, shall vest in and be removed and sold or disposed of by the Local Authority, and the proceeds thereof shall be applied in payment of the expenses incurred in the execution of the provisions of this section, and the surplus (if any) shall be paid on demand to the owner of the matter removed.

The expenses of removal by the Local Authority of any such accumulation, if and so far as they are not covered by the sale thereof, may be recovered by the Local Authority from the person to whom the same belongs, or from the occupier of the land, or where there is no occupier, from the owner.

PART IV.—DWELLINGS.

Subdivision I.—Houses Unfit for Occupation.

HOUSES UNFIT FOR OCCUPATION.

71. When it appears to the Local Authority that ANY HOUSE or OTHER STRUCTURE is unfit by reason of its FILTHY or DILAPIDATED condition or improper construction, or by reason of its being infected with infectious disease, or otherwise to be used or occupied, the Local Authority may give notice in writing to the OWNER OR OCCUPIER of such house or structure to PURIFY or REPAIR or ALTER the same so as to render it fit for use and occupation.

If the person to whom the notice is given fails to comply therewith within the time therein specified, he shall be liable to a daily penalty not exceeding ten shillings.

Moreover, the Local Authority may cause the house or structure to be PULLED DOWN OR DESTROYED, and may RECOVER from the person in default the expenses incurred by it in so doing.

FILTHY HOUSES.

72. When it appears to the Local Authority that ANY HOUSE or PART THEREOF is in such a FILTHY OR UNWHOLESOME CONDITION that the health of any person is, or is liable to be, affected or endangered thereby, or that the whitewashing, cleansing, or purifying of any house or part thereof would tend to prevent or check infectious disease, the Local Authority shall give notice in writing to the owner or occupier of such house or part thereof to WHITEWASH, CLEANSE, or PURIFY the same, as the case may require.

If the person to whom notice is so given fails to comply therewith within the time therein specified, he shall be liable to a DAILY PENALTY not exceeding TEN SHILLINGS.

The Local Authority may cause such house or part thereof to be whitewashed, cleansed, or purified, and in the meantime to be be vacated, and may RECOVER from the person in default the expenses incurred by it in so doing.

NUISANCES.

PART V.—NUISANCES AND OFFENSIVE TRADES.

Subdivision I.—Nuisances.

NUISANCES.

A nuisance is something which either actually injures, or is likely to injure, health, and admits of a remedy, either by the individual whose act or omission causes the nuisance, or by the Local Authority.

Per Lord Mansfield, C.J.—"To constitute a nuisance it is enough that the matter complained of renders the enjoyment of life and property uncomfortable."

79. (1) Any PREMISES in such a state as to be a nuisance or injurious to health; rat-runs on premises is a nuisance within the meaning of this section;

(2) Any POOL, DITCH, GUTTER, WATERCOURSE, or SANITARY CONVENIENCE, so foul or in such a state as to be a nuisance or injurious to health;

(3) Any ANIMAL so kept as to be a nuisance or injurious to health;

(4) Any ACCUMULATION OR DEPOSIT which is a nuisance or injurious to health; all "untidy" yards are dirty yards, and all dirty yards are a nuisance, within the meaning of this section;

(5) Any HOUSE OR PART OF A HOUSE so OVERCROWDED as to be dangerous or injurious to the health of the inmates, whether or not members of the same family;

(6) Any FACTORY, WORKSHOP, or WORKPLACE not kept in a cleanly state, or not ventilated in such a manner as to render harmless, as far as practicable, any gases, vapours, dust, or other impurities generated in the course of the work carried on therein, that are a nuisance or injurious to health, or so overcrowded while work is carried on as to be dangerous or injurious to the health of persons employed therein; and

(7) Any CHIMNEY (not being the chimney of a private dwelling-house) sending forth SMOKE in such a quantity as to be a nuisance; shall be deemed to be a NUISANCE LIABLE TO BE DEALT WITH IN MANNER PROVIDED BY THIS ACT.

NOTICE.

Your premises will be inspected by the Officers of the Local Authority and the Joint Epidemic Board, as well as by the Department of Public Health.

The co-operation of all householders, owners, or occupiers with the Health Authorities, is earnestly requested in enforcing the above provisions of the Act and ensuring the suppression of the PLAGUE.

Plague is a dirt disease, and is spread by Rats.

PREMISES INFESTED WITH RATS WILL BE DEALT WITH AS A NUISANCE.
KEEP RATS OUT OF DWELLINGS.

All fragments of food, vegetables, bones, &c., about dwellings and yards should be gathered up and placed in a securely covered rubbish-box.

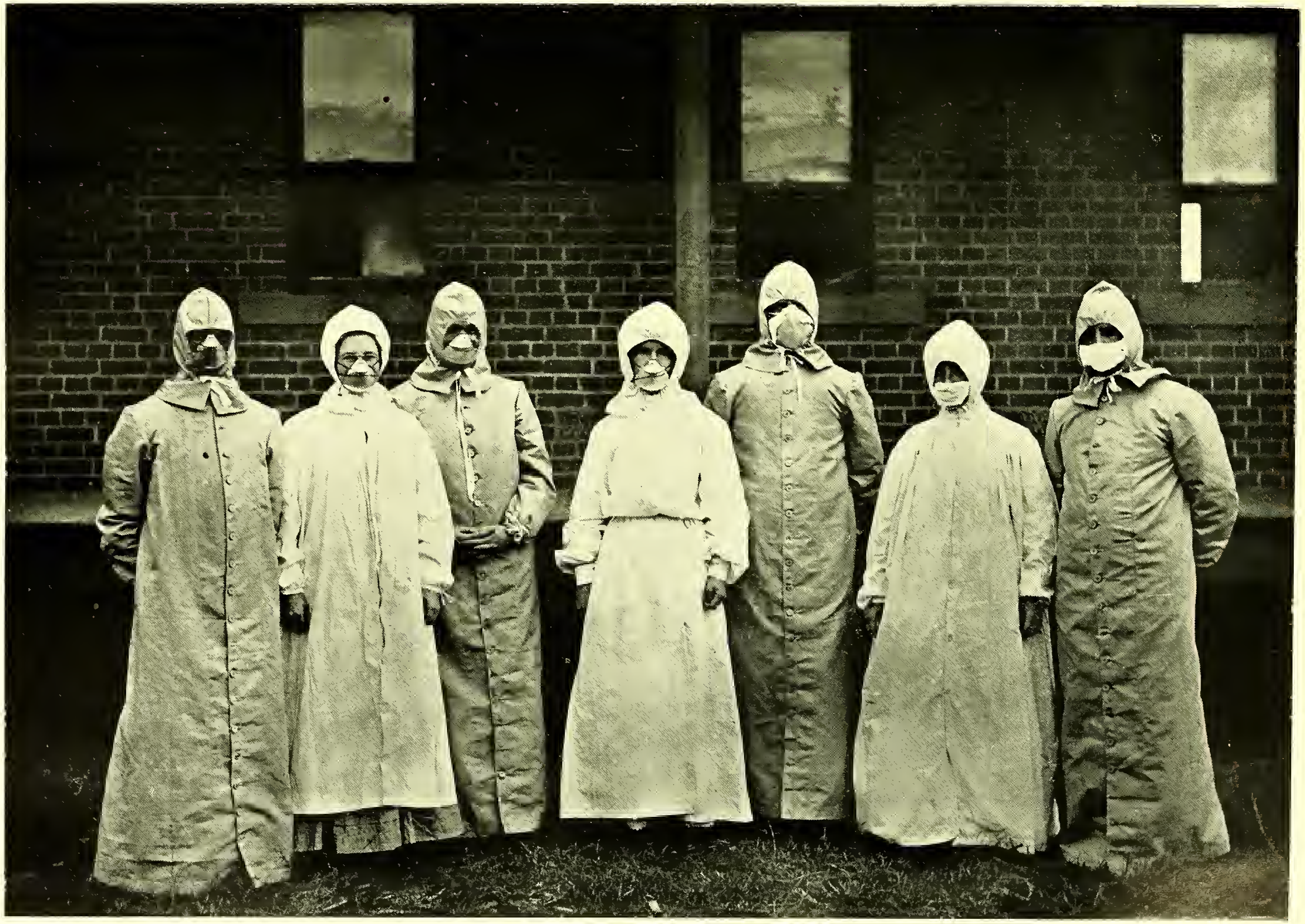
Every ratepayer should make a point of reporting every NUISANCE to the Clerk to the Local Authority.

NOTICE is hereby given that all OFFENDERS against the above provisions of "*The Health Act of* 1900," or the By-laws, will be PROSECUTED.

By order,

B. BURNETT HAM, M.D., D.P.H. (Camb.),
Commissioner of Public Health.

Maryborough Outbreak (Primary Pneumonic Plague), May-June, 1905.



Specially Devised Overalls and Respirators Worn by Dr. Burnett Ham and his Medical and Nursing Staffs.

PART IV.

Section I.

D.—THE EPIDEMIOLOGY OF PLAGUE.

I.—THE INFECTIVITY OF PLAGUE.

In the foregoing sections of this Report an effort has been made to set forth the main features of the disease, such as it would appear are essential to a proper appreciation of the facts which bear on the spread and communication of plague in Queensland.

The general question as to the relative importance of the various agencies which may have been operative in the spread of the disease, together with the epidemiological problems which naturally arise in connection with its mode of dissemination, have still to be discussed. A review of the evidence recorded above would appear to place beyond doubt the close association between plague in the rat and plague in man. This conclusion, it will thus be seen, has been founded on actual and extended observation. The strong character of the evidence upon which the plague rat stands condemned will appear on taking into consideration the now almost universal recognition of the danger incurred by its presence. Whether the Flea Theory or the Food Theory be accepted by authorities as best co-ordinating and harmonising all the observed phenomena with regard to the modes of dissemination of plague—the active part played by the flea in the transference of plague, or the ingestion of food contaminated with the plague virus—the rat must still be considered the *fons et origo* of infection in either case.

It is thus admitted by epidemiologists almost without exception that the rat is the principal agent in the dissemination of bubonic plague.

"The most conspicuous change in the opinion of experts in India regarding plague, since the issue of the resolution of 16th July, 1900, is the greatly increased importance now ascribed to the part played by rats in spreading and keeping alive the disease. . . . . In fact, almost all the evidence regarding the causation of plague may be regarded as pointing to the rat as the chief agent in its diffusion." (Resolution issued by the Government of India, Nos. 79-95, 17th January, 1906.)

The part played by the rat in the incidence, dissemination, and decline of epidemic plague was unanimously recognised by Australian observers from the first year of the introduction of the disease into Sydney and Brisbane in 1900. Every subsequent year has confirmed and added to the evidence collected and recorded by the health authorities in the capitals of both States.

Apart from the general evidence submitted, there are instances of a specific character which afford striking proof of the observed association of the epidemic with the epizoötic.

The small number of cases in connection with the outbreaks of plague in 1903 in Brisbane (19 cases connected with 15 places of infection) and 1904 in Sydney (12 cases with 9 places of infection) offered convenient opportunities for the observation and establishment of the most significant feature exhibited—viz., the presence of plague rats in every case at Sydney, and in all but two cases at Brisbane.

It is true that the mere proving of the presence of plague rats in a house or locality before human cases occur does not solve the problem of the dissemination of the disease by rats, but it would appear to offer *primâ facie* evidence as to the origin of the infection in man. In view of the fact that the infectivity of bubonic plague from man to man is practically nil, the occasional factor of man in the spread of plague need not be mentioned here, as a refutation of the statement with regard to the smallness of the risk incurred by direct contact, or close association, with plague patients taken generally. The epidemic in Queensland was not maintained by direct communication from the sick. The immunity of nurses, doctors, and others who were in attendance on the patient, and the small incidence on "contacts" and members of a household who were in close association with the attacked person, are instructive facts in this connection. With regard to the facilities offered for human inter-communication from infected to uninfected places, it cannot be denied that such communication could and actually did take place; but in no instance could it be shown that an "imported case" was the cause of an outbreak of plague in any part of the State. It has been commonly observed by Indian authorities that, while the rat was responsible for a local dissemination of plague, the spread of the disease from one locality to another was due directly to human agency. Human agency, apart from cases of pneumonic plague, as a factor in the infectivity of plague, is not within the experience of Australian observers, and, indeed, it is not necessary to assume that such human agency need occur; for it has been clearly demonstrated that where infected man could go infected rats could also and generally did go. The occasional instances in which secondary cases occurred in households were not, therefore, the result of close association with the sick, but a result from exposure to a source of infection common to all the inmates of the house—viz., the presence of infected rats. What was true of individual cases was equally true of certain groups of cases, living or working in localities which have been conveniently described as "infected areas." In these latter the infectiveness of the areas was entirely conditioned by the infectiveness of one or more of the buildings (generally a produce or other store offering special attraction for rats) which stood upon them.

Where several cases of plague occurred simultaneously, or within a short period of each other, the infectivity of the locality within which the attacked resided, or worked, cannot be inferred from the fact alone that the locality, or even infected premises within such locality, harboured plague-infected persons. Infectivity of houses, however, by reason of the presence of plague-infected rats within them, had here constituted a danger not only to the persons living in them, but also to others resorting to them.

The incidence on houses, even in "infected areas," was most erratic. It certainly could not be said in all instances that infected rats were found in all places where human cases occurred. In not an inconsiderable proportion of cases the common experience was to find carcases of rats, often too decomposed for definite decision as to plague-infection, either upon the infected premises themselves or upon other premises within a short distance of them. The incidence on members of a household was no less erratic than the incidence on premises. It was a common rule for only one member to be attacked, though all were equally exposed to infection. It will be seen, therefore, that infection was not conveyed by any of the methods common to other infectious diseases—influenza or dengue fever, for example—but that some special condition not commonly existant, some "chain of circumstances which is rarely complete in all its links, must be necessary," as Dr. Ashburton Thompson has tersely stated (Report on the outbreak of plague at Sydney, 1900, p. 36), "to the infection of man."

It cannot be denied that rats can infect their surroundings as well as man, and this capricious incidence exhibited by plague is better understood by the migratory habits of rats than by the more uniform movements of infected persons.

The dissemination of plague must imply a dissemination of the plague bacillus by some vehicle or by the movements of the hosts capable of carrying the germs. It also necessitates the more or less rapid passage of the plague organisms from one host to another. Where it could be shown in instances of this that non-communication from the sick to the healthy person was the rule, the appearance of secondary foci, yielding indigenous cases at some distance from the primary source or central focus of infection (*see* "Map B"), would tend to the conclusion that the infectiveness of localities is always due to the one and the same cause. Localities infected one year entirely escaped the next. Epidemic prevalence of plague in some towns of Northern Queensland occurred at one period to disappear altogether for several years in succession. It was also the exception for plague cases to recur in the same house in successive years.

Again, the disease was no more spread by mediate than it was by direct communication from the sick. We have no grounds for suspecting that either man, who presumably carried about infected clothing, or those who handled infected clothing, were important agents in the spread of plague. The incidence on nurses, doctors, disinfectors, and laundresses was so slight that it is hardly possible to believe that infection could have been communicated by means of infected clothing. As a matter of fact, but little opportunity was afforded for infection to have been carried in this way. As early as possible, usually within twelve hours of notification, the patient was removed to the Isolation Hospital; and the room, its contents, together with the personal effects of the patient, thoroughly disinfected and cleansed. Reference may be made here to a particular instance in which a straw mattress was thought to be the chief factor in the ætiology of three cases of bubonic plague during the outbreak of 1907, at Brisbane. A man, his son, and his daughter were in the habit of resting, during some portion of the day, on a bed containing the above-mentioned mattress. The bed was placed beneath the house—an old and dilapidated one built on piles 7 or 8 feet high—for the sake of the cool shade afforded. The front and one side of this underground room were formed by a rubble-stone wall, which afforded a perfect harbourage for rats. All three members of the household were removed to the Plague Hospital within a few days of each other. The man died, the boy and girl recovered. The burning of the mattress put an end to further cases among the remaining members of the family. But in this case, although mediate infection by a contaminated mattress might be suggested, the fact that this was literally swarming with fleas and bugs indicates that this explanation of the origin of plague in these successive instances of its occurrence is by no means an inevitable one.

In further consideration of the question of apparent mediate infection in merchandise and articles of commerce, it may be said that infection is conceivable when such commodities have been contaminated by the discharges of infected man or rats, or that it is inherent in goods likely to carry the latter.

In discussing the incidence on occupation, mention was made of the fact that produce and fodder, bags and bales, &c., were looked upon as "dangerous goods," in view of the heavy incidence of plague both in man and rats on the produce stores. The goods to which suspicion mostly attached were precisely those which proved most attractive to rats, either by the nature of the goods themselves or by reason of the places in which they were stored.

Observation has shown that cases in man occurred and plague-infected rats were found in previously uninfected places of the State, at precisely a time when fodder was imported from some place oversea, from a neighbouring State, or from local stores situated in an infected area of the metropolis; these places being one to which such merchandise had been conveyed.

The persons handling this fodder, or associated with the premises in which it was stored, were, in some instances, the only persons attacked. Cases at Ipswich, Bundaberg, Cairns, and Townsville, and a severe epizoötic among the rats at Childers, were thought to have originated by the introduction of presumably infected produce thereto.

This suggestion was, save in the case of two of these towns, quite unsupported by anything in the nature of positive evidence as to the actual conveyance of plague-infected rats with this fodder. At Ipswich and Bundaberg, the carcases of numerous plague rats were actually found among the produce stored in premises where the cases occurred. The heavy incidence of plague on produce-store employees, and to a less extent on grooms, jockeys, stablemen, drivers, and others whose duties included the feeding of horses, would seem to incriminate produce, grain, &c., as a source of infection. The latter were in many of the above instances traceable to stores to which infected rats had had access, but the discovery of rats, alive or dead, in this produce or in the bags, crates, or packing containing the same was quite an exceptional circumstance. That infection, in some way or another, may adhere to bags, fodder, produce, hay, &c., finds further support in the fact that specific cases can be cited to show that on four separate occasions packers of crockery, glassware, &c., in warehouses were attacked with plague after handling straw packing obtained from infected produce stores in the city of Brisbane.

It would seem, therefore, that while there are good grounds for believing that produce is responsible, but not necessarily *per se*, for the importation or carriage of plague in some instances, it is often difficult to obtain evidence of the conveyance of infection by fodder, grain, &c., even where such conveyance had actually taken place. The bacteriological data regarding the infectivity of merchandise is for the most part of a negative character. Rosenau (Bulletin No. 4, 1901, Hygienic Laboratory, Washington) has shown that the organism of plague usually dies in a few days on the surface of wood, paper, bone, and such-like articles of dry merchandise. He also found that clothing and bedding can harbour infection for a long time, and may act as fomites, and that food products, such as butter, milk, and cheese, may carry the infection of plague, but the bacillus usually dies quickly on the surface of fruits and prepared foods.

The suggestion that plague may have been imported into Australia in jute and jute bags manufactured in Bombay and Calcutta received support from the evidence of Dr. A. S. Lang, Chief Veterinary Surgeon and Bacteriologist to the French Government in New Caledonia. Dr. Lang, when on a visit to Brisbane in 1900, stated that he felt confident the outbreak of plague in Noumea was occasioned by the importation of large quantities of jute which has been shipped to Noumea from Bombay and Calcutta—at the time the latter were infected ports.

The Plague Regulations in force in Queensland and other States of the Commonwealth prohibit the importation of used produce sacks, and when it is realised that many of the new bags imported from India are made from material served out by the factories to natives who make it up in their own hovels where plague infection is rife, one can understand why this item of merchandise may well fall under suspicion.

Having now summarised what seems to us likely to be the positive evidence with regard to the introduction and spread of plague by means of merchandise, we may briefly consider the broader features of trade between infected and uninfected places. In the Report on the Indian Plague Commission (1898-99, vol. V., par. 262), it is stated: "The question of the possibility of plague being carried into countries oversea by infected merchandise is a very wide question, affecting, as it does, the general operations of trade. It would seem to us that the most important point to consider with regard to this question is the fact that, though plague might conceivably be conveyed in this manner, millions of tons of Indian merchandise have been imported into the great European ports, without giving origin to any outbreak of plague, either among the stevedores, who handled the cargo in bulk, or among the operatives who dealt in detail with the merchandise."

In the Thirty-third Annual Report of the Medical Officer of the Local Government Board, England (1903-1904), Dr. Theodore Thomson arrives at much the same conclusions. Dr. Thomson says: "In the earlier days of the present recurrence of plague in epidemic form, no measures whatever were taken as regards rats in our home ports; and, indeed, even to-day such measures are, in most of our ports, limited to ships known to have plague on board. Yet, notwithstanding our enormous mercantile traffic with all parts of the world, extension of plague on shore has taken place in four instances only—viz., twice at Glasgow, once at Liverpool, and once at Cardiff." It cannot be argued, however, that because the introduction of infection into countries oversea has not been directly traceable to merchandise, that the introduction of infection in certain articles of merchandise is impossible. It is difficult to understand that the virus, in the nature of the "naked" or unprotected plague organism, can remain latent in articles of merchandise for any lengthened period, and we have now reached a point at which it is appropriate to discuss how such infection may be artificially introduced. We have seen that sunlight, fresh air, and dessication will destroy the plague bacillus more or less rapidly, and that it does not survive long on the surface of inanimate objects exposed to the above natural agencies. That its growth, moreover, under conditions of external nature is restricted may be inferred again from the fact that competition with the ordinary saprophytic organisms in the struggle for existence would sooner or later put an end to its continued survival, for, as has been ascertained, its vitality even in pure culture, where no such competition exists, is often difficult to maintain. Indeed, it is owing to the more luxuriant growth of these common saprophytic bacteria that the existence or otherwise of the plague organism as a saprophyte itself cannot be satisfactorily determined in the soil or in the dirt of floors of infected houses.

The Report of the Indian Plague Commission (1901) records the fact that, in the very numerous series of experiments made in India, the plague bacillus has never been detected in the floors of infected houses by any trustworthy observer. The Indian Advisory Committee (1906) adds, "As far as we know this bacteriological feat still remains unaccomplished."

It would appear that the complex biological features of bacteria due to association and antagonism are as yet but little understood, the attempt to estimate the life history of the plague organism upon a variety of objects and under various conditions failing when a departure from natural conditions is made.

In order, therefore, to harmonise the bacteriological with the epidemiological observations, it must be assumed that the possible channels of infection are such as to secure for the plague organism a certain security against the bactericidal operations of Nature.

The Indian Advisory Committee (1906) concluded, as the outcome of elaborate experiments, "That animals can walk about and sleep on floors, which have been grossly contaminated with virulent cultures of *B. pestis*, without contracting plague, if only an interval of about twenty-four hours is left between the contamination and the placing of the animals on the floors." It significantly adds, however, that this obtained alone "as long as fleas were rigorously excluded."

The transport of plague-rats in fodder and other articles of merchandise must be regarded as of predominant importance where facts can be adduced to show that such conveyance has actually taken place. Infected rats can and do get ashore from ships, in cargo and other ways, in spite of the utmost vigilance, and in this connection the initial source of infection may ofttimes be unobserved.

That the health authorities are cognisant of such danger from rats is evidenced by the elaborate precautions adopted at each port to prevent the access of ship-rats to the shore or shore rats to the ships.

While the plague-rat is a necessary factor in the spread of plague, it does not follow that actual contact with the plague-rat is necessary to or even determines the infection of man. It has already been shown that where such contact occurred infection was the exception and not the rule. It will also be shown presently that in several places abroad epidemics of plague have occurred without a corresponding epizoötic amongst rats; and, on the other hand, an outbreak amongst rats at Childers, in extra-coastal Queensland, ran its course and terminated with but one single case in human beings. In discussing the geographical distribution of rats, particular attention will be called to the species of rats and, when considering the subject of plague and rat parasites, the species of fleas which, in our opinion, are more directly concerned in the spread of epidemic plague. To revert, however, to the conveyance of plague by particular articles of merchandise. Apart from the conveyance of plague-rats in produce and other fodder likely to attract rats, the operation of some other mode of conveyance of infection, such as the carriage of plague-infected rat-fleas in the incriminated articles, would throw much light on many hitherto obscure features in connection with the question as to whether merchandise is to be regarded as infective. This extension of the rat-flea hypothesis would explain why bags, bales, clothing, grain, produce, and other fodder could carry infection in the absence of plague-rats. It also affords an explanation of the infectivity of houses into which infection has been introduced by rats, the fleas remaining when the rats have fled or died.

Apparent persistency of plague in soil (soil infection) and the "clinging" of infection to certain tenements, in the absence of a continuance of rat presence, is also similarly explained by a constancy in flea occurrence. Apart, indeed, from this, there is no evidence with respect to Australian occurrences of "place infection."

In connection with the storage of grain and fodder, it is a well known fact that fleas often swarm in the dust and *débris* of the chaff, &c., on the floors, wooden or earthen, of the granaries. If old and stale-looking chaff, which has lain for some time in dark and dirty places, be carefully sifted, fleas will often be plentifully found in it. The common flea on the domestic rat, *M. rattus*, is the *Pulex cheopis*. This flea is sensitive to light, and lives with its host in dark places among the produce bags and in the roofs of houses. It has been recorded by trustworthy observers that each flea has its definite host, and, under ordinary circumstances, will stick to its host; but, when driven by hunger, in the absence, or on the death, of its host, it will attack man. That this flea is an important agent in the transference of plague from rat to rat is evident from the important experiments recently carried out by the Indian Plague Commission (1906), but whether the *P. cheopis* plays the same active part in the transfer of the disease from rat to man no direct experimental evidence can, of course, be forthcoming. The possibility of infecting other Primates by rat-fleas has, however, been shown by the Indian Advisory Committee, monkeys being the object of experiment (*vid.* p. 158). The fact of contact infection arising from propinquity of man to man being so little in evidence receives an explanation in the fact that, notwithstanding the passage of a flea from its proper host to man, or *vice versâ*, evidently takes place, the passage of the rat-flea from man to man (man being only a facultative host) must be a comparatively rare contingency. The reports on plague investigations in India, issued by the Advisory Committee, however, show that *P. cheopis* will not only under certain circumstances readily bite and feed on man, but, also, that this species of flea can be kept alive for more than three weeks by feeding it on man alone.

In connection with the importation of trade under circumstances which exclude the intervention of rats, the question which now presents itself for solution is the length of time the plague-infected rat-flea will live in the particular environment into which it may in such case be introduced in the absence of its host? The infectivity of clothing and such articles may, perhaps, receive its explanation from the fact that fleas that have partaken of a meal will persist for a long time in the shelter that these objects are capable of affording. This explanation has been thus alluded to by Bannerman: "It seems (he writes) to explain the infectivity of clothes so often noticed. It is probably the fleas in the clothes that are the danger not the clothes themselves."—("Journal Hygiene," April, 1902.) We have seen that for the transference or transportation of the plague bacillus some security for the bacillus, such as is found in the body of the flea, is probably necessary during its passage from host to host. The flea has great powers of endurance, apart from its host, and comparative longevity. The rat being the proper host of the rat-flea, the fact of its attacking man—and it is abundantly evident that it will do so—is fortunately an incident in its economy compelled by exceptional circumstances—its deprivation by an emergency of its host, the rat. If we assume that the spread of plague depends on a dual process of adaptation on the part of the plague bacillus to growth within the rat or human organism on the one hand, and to survival in a more or less virulent condition within the body of the flea or other insect on the other hand, the question would become one of the greatest importance in connection with the epidemiology of plague, if it could be shown that the plague bacillus undergoes a numerical development in the body of the flea. As to the latter hypothesis, that the flea is a real intermediary host of plague, evidence is not wanting. The Indian Plague Commission has recently made a large series of observations to determine the presence or absence of abundant plague bacilli in the stomach contents of fleas, and established the conclusion that "multiplication of plague bacilli may take place in the flea's stomach."—("The Journal of Hygiene," Vol. 7, No. 3, July, 1907, p. 4.)

If the flea is a mere passive carrier of the plague organism, why not the bug and the mosquito? In rats dying of septicæmic plague, the blood of the circulation contains the *B. pestis* in enormous numbers, which could readily be abstracted by any blood-suctorial insect. If the flea be an accidental agent in the dissemination of the disease, the infective material is probably carried on the feet lancets or other portion of its anatomy, and flies beetles and cockroaches having access to contaminated foodstuffs could probably act in the same way. With regard to cockroaches alone, have we evidence to show that infection can be thus communicated from infected to healthy rats, but it is doubtful whether any great importance can be attached to this isolated observation, as indicating a method of transmission in the epizoötic or epidemic spread of plague.

That the virus may effect an entrance into the animal organism through the channel of the alimentary canal is evidenced by the facility with which animals take plague by feeding. The suggestion that man not unfrequently contracts the disease by the ingestion of plague-infected food contaminated by an infected rat is for the most part based on the results obtained by feeding animals with pure cultures, with tissues containing the plague organisms, or with plague material from man, rats, or other animals. The *post-mortem* examination of rats fed with plague material revealed the presence of definite and well-marked pathological changes in the intestines. Hunter, of Hong Kong, called attention to the great frequency with which the intestines were the seat of primary hæmorrhagic lesions in a large number of autopsies conducted by him on plague cases, and in his "Report on a Research into Epizoötic and Epidemic Plague," 1904, he has given a detailed description of these pathological changes met with in the alimentary canal and mesenteric glands. Simpson (1905) confirmed the pathological description given by Hunter.

Klein (The Bacteriology and Etiology of Oriental Plague, 1906) records a considerable number of feeding experiments in which he was successful in giving the disease to rats, mice, and guinea-pigs by protecting the cultures and organs of plague-infected animals by first drying them along with the food, or by administering the virus with the food in such a state that part of it was not affected by the gastric juice of the stomach. He found that the plague bacilli taken in the contaminated food in this way multiplied in the intestines, entered the lacteals of the latter, and so invaded the blood stream in considerable numbers. The infected animals died between the fourth and sixth days. In every instance enlargement and congestion of the mesenteric glands, with definite pathological changes in the intestines, were present. Klein says: "The positive results obtained by feeding with semi-dried or more completely dried gelatinous plague materials—*e.g.*, gelatine cultures and plague organs dried by themselves or mixed with foodstuffs (wheat, rice, flour)—are in marked contrast to the negative results of feeding with fresh plague materials—*e.g*, fresh milk cultures, broth cultures, watery emulsions of cultures of *B. pestis* and fresh plague organs."

The above facts would certainly support the view that plague may effect an entrance into the animal organism by the digestive tract, and that a similar condition might occur in respect of human beings, the point of infection, as Klein states, being chiefly the intestine itself.

The results of laboratory experiments, however, cannot altogether be received as indicating an actual approximation to natural conditions. This has already been mentioned in connection with the use of the Danysz rat virus as a method of destroying rats. The actual results achieved in the open air were not at all proportionate to the results obtained with laboratory animals. The virus in the latter case was uniformly kept at and administered in the highest state of its activity and vitality, and there was little chance for contamination by saprophytic bacteria, or of the various modes of escape which must always be operative in Nature.

The Indian Plague Commission ("Journal of Hygiene," July, 1907) directs special attention to the *post-mortem* appearances of rats infected by feeding and those found in rats naturally infected. In the case of fed rats, the intestines were congested with hæmorrhages and ulceration of Peyer's patches, and the common site of the bubo was the mesentery. In naturally infected rats, the stomach and intestines showed no subcutaneous hæmorrhages and no mesenteric bubo had been observed in 5,000 *post-mortems*. Moreover, the pathological changes corresponded to those exhibited by rats that had died of plague under artificial conditions, in which the disease was due to inoculation—flea-bite. These observations afford very striking evidence that rats in Nature are not infected by feeding. Both Simpson and Hunter have contended that the ingestion of food contaminated with the plague virus is the cause of the septicæmic form of plague in man. This theory was formulated by them on their observations in Hong Kong of *post-mortem* examinations of plague cases and experiments on wild rats fed with fresh plague organs. The pathological changes described by these observers are, however, practically present in every septicæmic case of plague, whether following on the ordinary bubonic type or occurring primarily as a septicæmia without buboes. In many of our own cases, in which infection was thought to have been caused by ingestion of contaminated food, the characteristic *post-mortem* appearance of engorgement and hæmorrhage was frequently associated with enlargement and inflammation of the retro-peritoneal glands, and not infrequently of the mesenteric glands.

Again, both rats and mice commonly die of the septicæmic form of plague whether caused by ingestion or inoculation. Judged by the pathological changes observed in the dead bodies of human beings, as hitherto recorded, it would appear that more evidence and further observation are necessary before any definite pronouncement can be made with regard to Dr. Simpson's contention that "septicæmic plague is in most cases contracted by the alimentary canal." At best, however, the food theory does not explain epidemic plague.

II.—THE RELATION OF THE EPIZOOTIC TO THE EPIDEMIC.

The observation that rat mortality preceded or accompanied certain epidemics of plague is a very ancient one. The *recognition* that rats died of a disease—caused by an organism morphologically and culturally identical with a microbe recovered from the tissues of human beings attacked at or about the same time—and recognised as plague, was, however, a phenomenon of comparatively modern date. The two important factors which had to be determined in connection with the epizoötic were—firstly, that the actual disease attacking rats and affecting human beings was undoubtedly plague; and, secondly, that the epizoötic had a relationship with plague in man with regard to place and time. That rats constitute the chief, if not the sole, source for the bubonic infection of man is a statement that has now been freely conceded by many observers, though denied by some.

Taken as a bald statement of fact and without reference to a reasonable causality as between rat and man, it must be admitted that it falls short of the importance to which Australian observers, at any rate, attribute to it.

Apart from the evidence of the observed association, the question of causality has hitherto been left so open a question by Indian and other observers that, not having acquired the data necessary for the decision of the question, they have fallen back on a negative line of argument.

There is a difference between the observation of rat plague and the identification of rat plague. Only a continuous and systematic investigation of the rat all the year round, in the period of freedom of plague as well as in the period of prevalence, can establish infection of the rat as a necessary factor in the infection of man.

The contentions that but little is really known of many bacillary diseases outside the human organism; that rat and man may take plague from some common source at present unknown; that an epidemic of plague may occur without a coincident epizoötic amongst rats; that actual contact with the plague rat is unnecessary to infection of man; that plague rats are often found on premises unattended with cases in man, &c., would only apparently sustain a correct judgment regarding this question concerning the association of epidemic and epizoötic plague in so much as the observations relate to a more or less short period of observation and a narrower field of operation.

The anomalous character of the above observations may possibly be explained by the fact that, while the plague rat is directly involved in the dissemination of the infection, the plague rat is not the only factor concerned. We do not contend that it is.

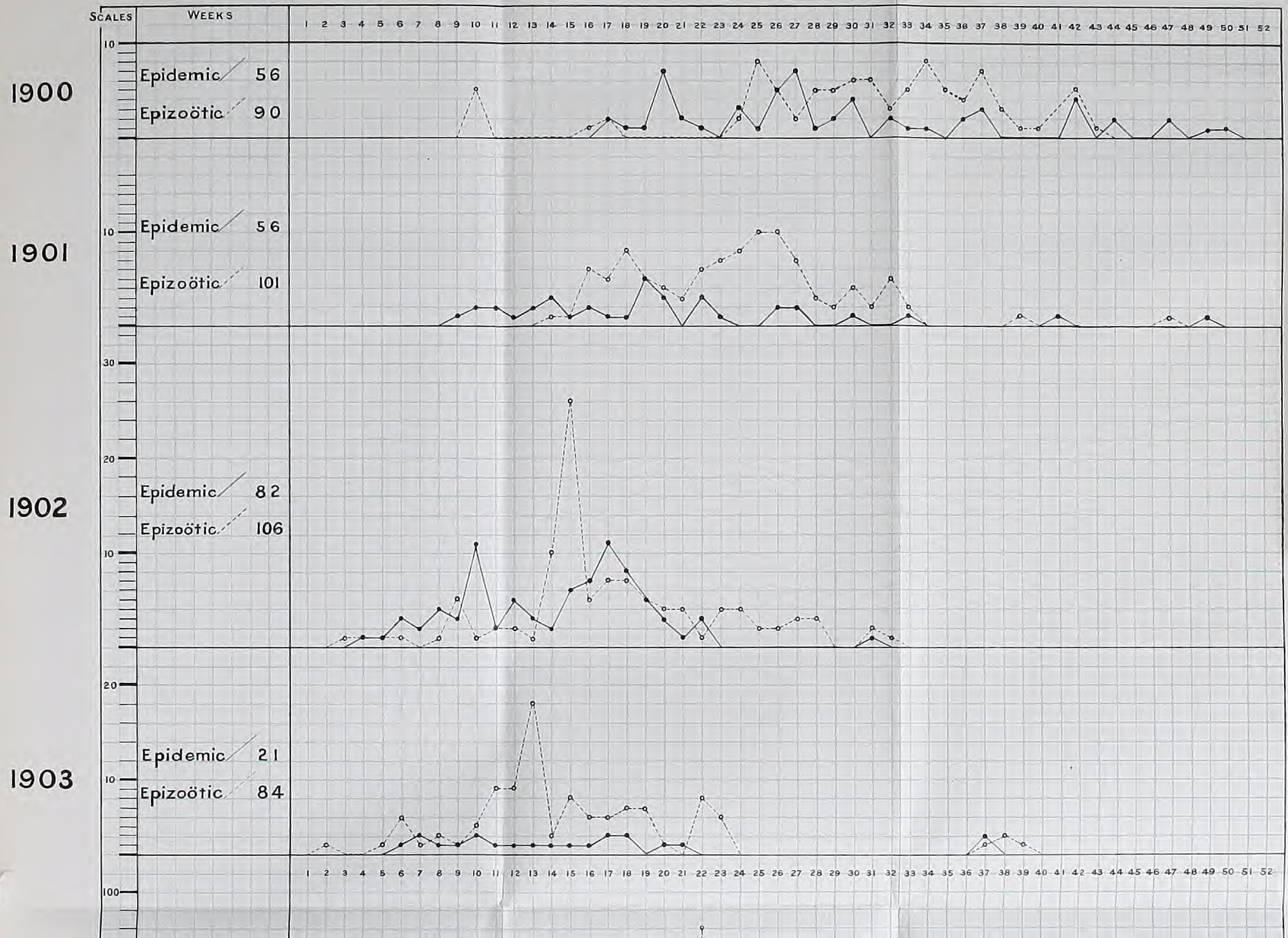
An intermediary between the rat and man is necessary.

That the epizoötic (Rat Plague) and the epidemic (Human Plague) are commonly successive is a fact in harmony with the observation that when any animal succumbs or, indeed, is even moribund from one cause or another the fleas that have been subsisting on it leave it and attach themselves to another animal host, should there be one that can afford them sustenance, and we may thus on occasion observe the coexistence of rats dead of plague, fleas, and human plague.

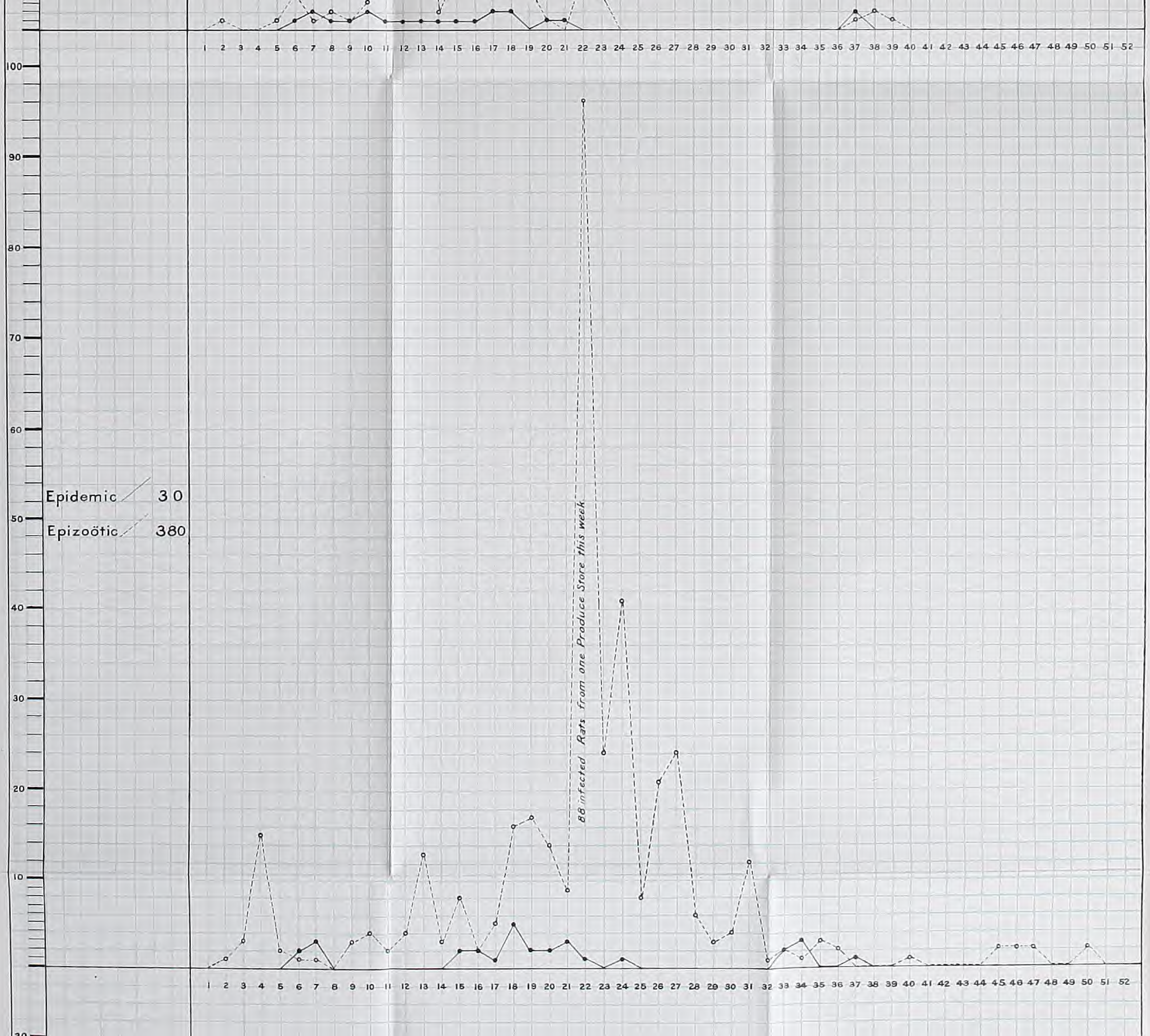
Rat mortality was invariably discovered in every indigenous centre of plague throughout the State, the rise in rat infection always preceding the manifestation of the cases in man. Epizoötics occurred at periods when epidemics were prevalent, and both took place at certain seasons of the year which extended observation led us to anticipate. The association was observed year after year. Sporadic cases in man during periods of quiescence were also attended by the presence of plague of sporadic occurrence in rats. The assumption that the epidemic was a consequence of the epizoötic is further based on the fact that the dissemination of travelling plague cultures in the living bodies of rats was over areas which coincided with areas over which the epidemic was observed to extend. A survey of the charts appended hereto will show the weekly course of the epidemics and epizoötics for the years 1900-1907 within the Metropolitan Area.

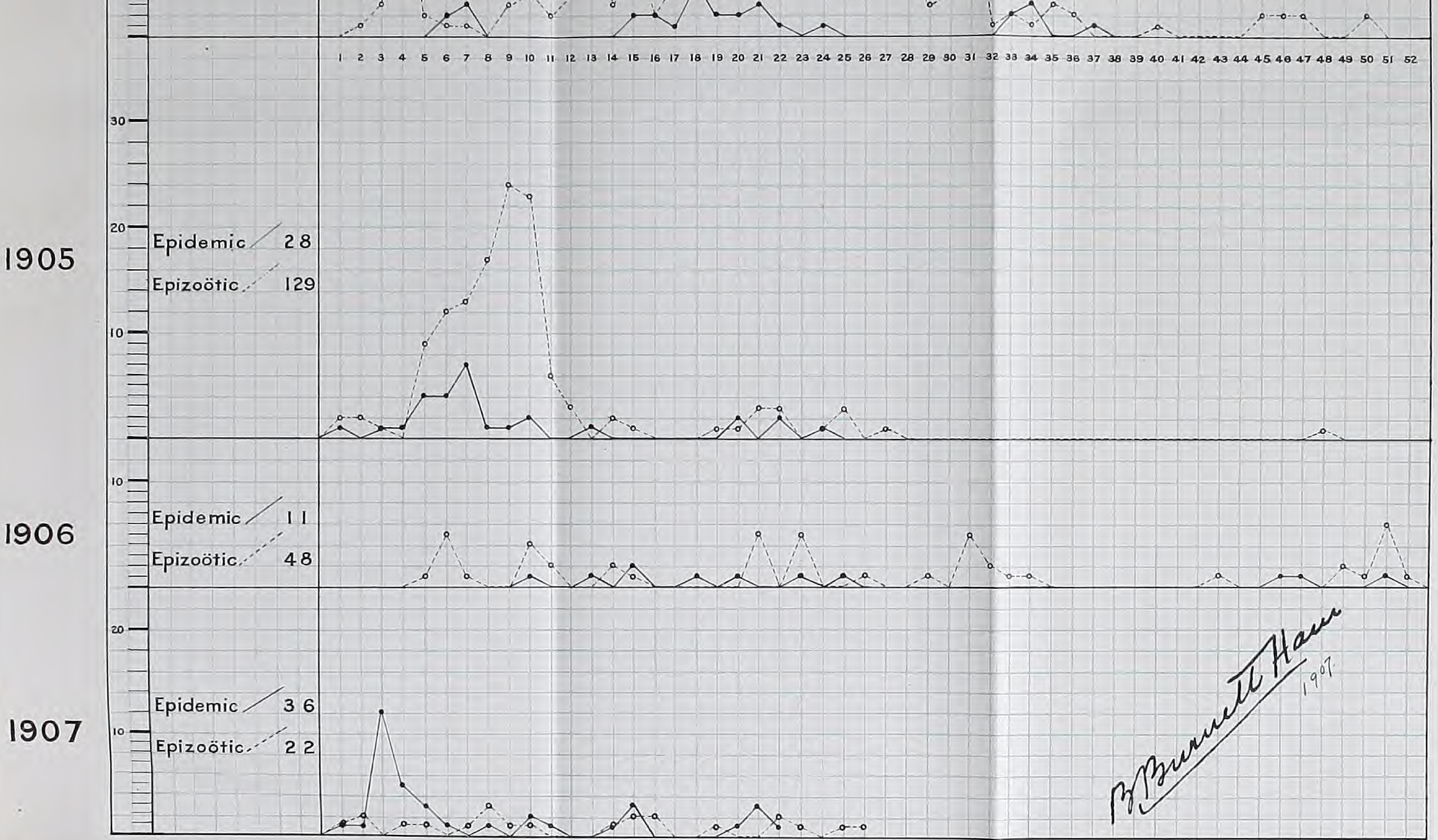
CHART SHEWING THE WEEKLY COURSE OF THE EPIDEMIC AND EPIZOÖTIC

— FOR YEARS 1900-1907. —



1904


Epidemic 30
Epizoötic 380
100
90
80
70
60
50
40
30
20
10
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48 49 50 51 52
88 infected Rats from one Produce Store this week



B Burnett Ham
1907.

The epidemic is marked by a continuous line, the epizoötic by a dotted one.

In connection with the epizoötic curves it may be explained that they only indicate approximately the proportional severity of the disease amongst rats, since the number actually found infected in the daily collection made for bacteriological examination must fall far short of the number naturally infected in the open, but, for obvious reasons, not obtainable. The rat curves, therefore, indicate the extent and result of the search, but cannot be considered as complete. It will be noticed for the years 1900, 1902, 1903, 1904, and 1906 that infected rats were discovered previous to the cases in human beings.

In 1901, 1905, and 1907, human cases apparently occurred early in these years, either before, or concurrently with, the mortality of rats; but on reference to the charts it will be seen that infected rats were found in the latter end of the year—November and December—immediately preceding those under discussion.

The conclusions deduced from a study of the charts are the following:—

1. That a close association exists between plague in rats and plague in man.
2. That the epizoötic invariably precedes the epidemic.
3. That the epizoötic runs concurrently with and outlasts the epidemic.
4. That an interval, usually short, elapses between the death of rats and the attacks of human beings.
5. That the curves of the epizoötics bear a general resemblance to the curves of the epidemics, the rise and fall in the former being followed by a rise and fall in the latter.
6. That both the epidemic and the epizoötic exhibit a period of incidence, stasis, and decline.
7. That the incidence on both rats and man is heaviest in the months of April, May, and June.
8. That from the middle of the year—end of June—to the end of the year, there is a marked period of quiescence.
9. That in a few years this period of freedom is broken by sporadic cases both in man and rats.
10. That both the epidemics and the epizoötics have been of comparatively modest dimensions.

III.—SPECIES OF RATS.

A knowledge of the life history, habits, distribution, and habitats of the various species of rats is essential to the proper study of plague.

In Brisbane, at least five kinds of rats have been met with, but, practically speaking, there are only two distinct species of rats with which we are concerned in Australia—viz., the *Mus decumanus* and *Mus rattus*.

1.—The *Mus decumanus:* The common Grey, Brown, or Norway Rat.

Chief features—Body: Thick set, strong, and muscular; claws blunt for burrowing with. Head: Face broad with prominent black eyes; short round blunt ears covered with very fine hair; blunt nose; short and chisel-shaped incisors. Tail: Thick, shorter than head and body combined, and about same length as the animal's body; blackish in colour, lighter below, and blunt at the tip.

Habits—Haunts sewers, drains, cellars, and basements. Feeds on garbage, and has great cannibalistic propensities. Does not live in captivity.

2.—The *Mus rattus:* The Black or Old English Rat.

Chief features—Body: About two-thirds the size of the *Mus decumanus;* long and slim; has a bright, blackish-ruddy coat; claws sharp for climbing. Head: Face sharp, with pointed nose; eyes bright and prominent; ears oval, long, delicate, and almost transparent, with very few hairs on them; incisor teeth sharp and pointed. Tail: Long and slender, equal in length to the body plus the head; distinctly blackish in colour and pointed at the tip.

Habits—Is a nimble climber. Lives in the house, in ceilings, upper floors, and rooms; also lives in trees. Feeds on grain, fruit, and the household foodstuffs. The ship rat. It is easily tamed, but will not breed in captivity.

3.—*Mus Alexandrinus rufus:* This rat is not a distinct species, but a variety of the *Mus rattus*, or European black rat. It has a rufous or ruddy-grey coloured coat intermingled with a few black hairs. In size, appearance, and habits it simulates the *M. rattus*. It has long thin ears, and a tail longer than the head and body combined, and tapering to a point; blackish in colour. Has sharp claws for climbing. It is also a house rat, but does not live in trees so frequently as the *M. rattus*.

4.—*A Hybrid Variety:* This rat is probably a cross between the *M. decumanus* and *M. rattus*. Has small ears, long slender tail, and a reddish-brown or grey coat. In size it is between the grey and the black rat, but larger than the *M. Alexandrinus*.

5.—*Hydromys leucogaster:* The common water rat. It is about twice the size of the grey rat, and has a broad head with small ears; a thick tail, which about an inch from the end is covered with long white hair. Unlike the other Muridæ mentioned that are naturalised species, this is an Australian indigenous rat.

6.—*M. musculus:* The grey mouse.

From the above descriptions it will be seen that the habits and modes of life of the two more important rats—viz., the *M. decumanus* and the *M. rattus*—are totally different. The grey rat (*M. decumanus*) is a burrowing rodent. It is the "sewer" rat, and lives apart from man in sewers, drains, outhouses, rubble-stone walls, burrows, &c. It is an extremely aggressive animal, as will appear from its cannibalistic propensities already mentioned.

It is an interesting fact that this rat has driven away the *M. rattus* from Great Britain and other European countries wherever these two species of rats have come in contact.

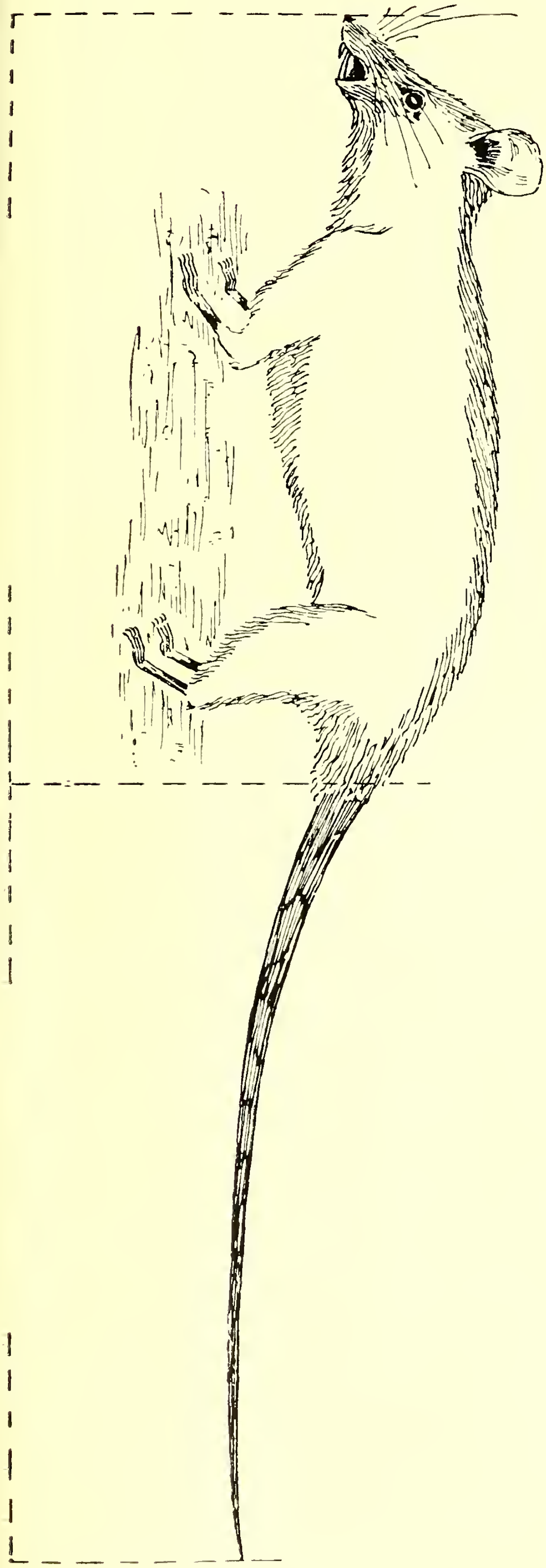
The *Mus rattus*, together with its variety, *M. Alexandrinus*, is a cleaner, smarter-looking animal than the grey rat, and much more active in its habits. It is now practically extinct in Great Britain, having been ousted by the grosser and more ferocious *M. decumanus*. It lives in the house with man, generally in the ceilings and roofs, and in the interspaces between the external and internal walls of wooden tenements. Man provides it with both shelter and food. It does not burrow, but has distinct climbing propensities, and may be often seen among the branches of trees and bushes.

The agility of these rats is well exemplified when one happens to be enclosed in the room of a house during search operations. It will climb the legs of tables and chairs, and jump from one picture frame to another as nimbly as would an opossum. On the other hand, the *M. decumanus* is content with roaming round the floor and trying to get behind any obstacle which may hide it from view. It is stated on good authority that the black rat (*M. rattus*) disappeared from Europe about the same time as did the plague.

The enforced separation of the house or black rat from man—by reason of the better housing of the latter, and the advance made in sanitation—since the great epidemics of plague in Europe, must necessarily have interfered with its habits, and so rendered it an easier prey to the more warlike grey rat dwelling in the sewers or in the open.

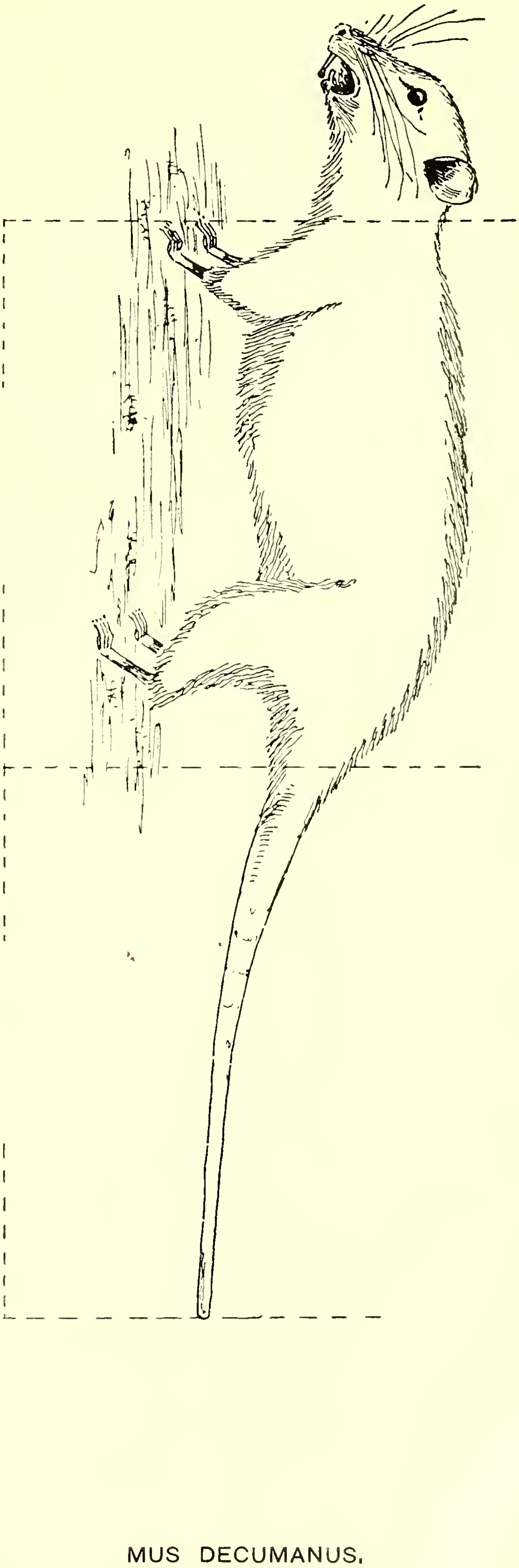
Of the imported varieties of rats in Brisbane, the *M. decumanus* predominates; the *M. rattus* comes next, and the *M. Alexandrinus* last. All the above three rats are susceptible to plague, and can, by themselves, cause plague in man. The *M. musculus*, the grey mouse, is less susceptible to plague than are rats, and the number found plague-infected was comparatively small.

The hybrid variety of rat mentioned above has also been found susceptible to experimental and natural plague-infection. The disease has also been induced in each of the species mentioned above, both by feeding and direct subcutaneous inoculation with cultures, with lymph, or with blood from a naturally or experimentally diseased animal. Practically, there was no difference as to the susceptibility of any one kind of rat, with the exception of the large water rat and the bush rat, neither of which last-mentioned species have been found susceptible in nature, although both these species have occasionally been found associated with infected grey or black rats on wharves and in warehouses situated near the river frontage.



MUS RATTUS.

Colour: Black, or reddish-black, white below.
Head: Nose long and pointed.
Ears: Large and thin.
Teeth: Sharp and pointed.
Tail: Thin, equal in length to body plus head, distinctly black in colour, and pointed at tip.
Claws: Sharp.



MUS DECUMANUS.

Colour: Brown or grey.
Head: Nose short and blunt.
Ears: Short and thick.
Teeth: Blunt.
Tail: Equal in length to body minus the head, brownish in colour, lighter below, blunt at tip.
Claws: Blunt.

In fully three-fourths of the infected rats the disease was septicæmic or generalised, *B. pestis* being found in all the internal organs; while in the remaining fourth the infection was localised in the lymphatic glands of either the femoral or the axillary regions.

It was further ascertained by a series of experiments conducted at the Bacteriological Institute, by Mr. C. J. Pound, that little or no difference existed in the degree of immunity in any of the susceptible species when at one time rats of about the same age were chosen as subjects for experiment, the very young of the susceptible species being more readily infected when inoculated with an attenuated culture which older animals of the same species were able to resist.

At Brisbane it has been ascertained that rats breed all the year round with but little variation in any particular month.

IV.—THE DISTRIBUTION OF RATS AND RAT-PLAGUE.

The comparative immunity of places where cases of bubonic plague have occurred unaccompanied by rat-infection has been the subject of much speculation by observers as to the probable cause.

A series of specific instances can be mentioned to show, on the one hand, that in some infected places plague in human beings made its appearance in the absence of rat-mortality; and, on the other hand, that plague occurred among shore-rats, unaccompanied by any, or, at most, one or two, cases in man (*vid.* p. 131-2).

In Australia, plague cases were reported at Perth and Fremantle, in West Australia, during the years 1900 and 1901. Rat plague was also observed in these towns.

One or two sporadic cases of plague in man have been recorded in Melbourne, Victoria; and in Adelaide, South Australia; since the year 1900. It is, however, only at Sydney and at Brisbane that plague in epidemic and epizoötic characters has been manifested to any degree.

The immunity enjoyed by Victoria, South Australia, and Tasmania has been so thoroughly recognised for the past eight years, that any cause which may probably explain this limitation of the disease would throw some light on a most important factor in the epidemic occurrence of plague. The suggestion that preventive measures have been more efficiently and thoroughly planned and carried out at Melbourne, Adelaide, and Hobart, can at once be dismissed, in view of the fact that the Plague Regulations, first formulated by the Australian and Tasmanian Intercolonial Plague Conference of 1900, are practically enforced in every State of the Commonwealth. Indeed, the opportunities afforded the New South Wales and Queensland Health Authorities—by reason of the numerous epidemics and epizoötics which have prevailed in these States, and the sanitary activity stimulated by the same—have enabled them not only to advance knowledge of the mode of spread of plague, and to gain much practical experience in the methods of prevention, but also to inform themselves of many sanitary and essential requirements not formerly considered or heretofore correctly estimated. Neither can it be maintained that the general sanitary conditions of the more Southern capitals of the States of the Commonwealth are so superior to either Sydney or Brisbane as to ensure a continued and effective protection.

Under the panic of the earlier epidemics, and under the influence of a reasonable desire to adopt measures for averting a future or controlling a present epidemic, the resources at the disposal of the Governments and the municipal authorities of New South Wales and Queensland have been employed to limit the extension of the disease by every practicable means. The importance of effecting at least the essential sanitary reforms in the more Northern as well as in the Southern capitals is apparent when it is realised that "no place, however remote from centres of infection, can be regarded as protected by its position."

The danger of introducing infection by plague-stricken rats, or by infective produce and merchandise, is at least as great to the southern ports as it is to the northern ports of the Commonwealth.

A more feasible explanation of the immunity of the more southern states is, perhaps, one based on the distribution of the species of rats, and the prevalence of particular fleas found on them.

It can hardly be supposed that the absence of noticeable rat-mortality in an epidemic of any proportion can be attributed to the scarcity of rats in the localities in question. The Indian Plague Commission of 1898-99, in commenting on this particular point, directs attention to the fact that "it is one of the as yet unsolved problems, in connection with the epidemiology of plague, why the disease has spread in different degrees among rats in different localities and different seasons."

In discussing the species of rats found in Brisbane, mention was made of the fact that the *Mus rattus*, from its habits of breeding and nesting in the roofs of houses with man, was much more likely to infect man through its ecto-parasites than the *Mus decumanus*, which shuns man as far as possible, and from its habits of living in sewers and drains is not so likely to come into intimate contact with him.

This modified rat-flea theory has been based upon, and supported by, many incidents in the history of the present pandemic of plague.

Captain W. Glen-Liston, I.M.S., now a member of the Indian Plague Advisory Committee, called attention to the habits and distribution of the *M. rattus* and *M. decumanus* in an interesting article published in "The Indian Medical Gazette," 1905. This observer also in 1904 showed that in India there was one flea (*P. cheopis*) that infested rats more commonly than did another; that this flea might harbour plague bacilli in its body, and that this special rat-flea was one that would bite man. Liston says, "These fleas will take to an animal which is not their normal host. Some of these fleas have been shown to be infected with large numbers of plague germs in their stomachs, and these germs, far from being destroyed by the digestive juices of the stomach, seemed to be multiplying and in a healthy state."

Some illustrative facts upon which our opinion has been based are the following:—

The *M. rattus*, formerly the common rat of England, has almost entirely been displaced in Europe generally by the *M. decumanus*.

The *M. rattus* (or its variety, the *Alexandrinus*) is the common domestic rat of India, though the brown rat (*M. decumanus*) does also occur.

In South Africa, the *M. rattus* is the prevailing rat.

In Australia, the *M. decumanus* is the commoner rat, though the *M. rattus* is largely in evidence.

At Sydney, Newcastle, and the Ulmarra district in New South Wales, the grey rat (*M. decumanus*) is in a decided majority. The *M. rattus*, however, has been found infected in comparatively large proportions in these places, considering the number of the former as compared to that of the latter collected for examination.

The same may be said of Brisbane and Rockhampton, in Queensland; the number of *M. decumanus* examined being in proportion of about 4 to 1 of the *M. rattus*.

In Townsville and Cairns, however, where epidemics and epizoötics have been of frequent occurrence, the *M. rattus* (or *Alexandrinus*) is the more common rat of the two.

At Melbourne, Victoria, the *M. decumanus* is practically the only rat found in the city itself, a few *M. rattus* being occasionally found about the wharves and shipping.

No epizoötic has, as yet, been recorded in Melbourne.

In West Australia (Perth and Fremantle), where plague in man and rats has from time to time occurred, the *M. rattus* is the commoner rodent met with.

In Tasmania, both species occur, but the comparative proportion has been undetermined.

From the above list it will be seen that the *M. rattus* is found in places where epidemics and epizoötics of plague have been observed—viz., India, South Africa, Sydney, Newcastle, Ulmarra, Brisbane, Townsville, and Cairns.

In the majority of these places, the *M. rattus* has been largely found infected, and, while it is true that the *M. decumanus* has been apparently more directly implicated in Sydney and Brisbane as the infected rodent, a glance at the table below will show that, in proportion to the numbers of the two species examined, the incidence of the disease is really heavier on the *M. rattus* than on the *M. decumanus*.

This is especially noticeable in the figures given below for the year **1906**. Of 9,275 *M. decumanus* examined at Sydney, 46 were found infected; of 8,694 *M. rattus* examined, 89 were plague-infected. For the same period in Brisbane, 16 of the rats examined and found infected were *Mus decumanus*, while 32 were of the *M. rattus* species.

The following table has been kindly supplied to me by Dr. Ashburton Thompson, Chief Medical Officer of the Government of New South Wales:—

Table showing Number and Species of Rodents Examined at Sydney for Plague, and Number found Infected during Years 1904-1906.

Species.	1904.		1905.						1906.	
	Sydney.		Sydney.		Newcastle.		Ulmarra District, Clarence River, N.S.W.		Sydney.	
	Examined.	Infected.	Examined.	Infected.	Examined.	Infected.	Examined.	Infected.	Examined.	Infected.
Mus decumanus ...	18,442	108	11,383	78	4,438	171	1,848	106	9,275	46
Mus rattus ...	7,318	73	5,681	45	1,477	13	119	1	8,694	89
Mus musculus ...	26,254	62	14,831	18	738	22	716	3	11,478	39
Total ...	52,014	243	31,895	141	6,653	206	2,683	110	29,447	174

The following table gives the figures for Brisbane for the same period:—

BRISBANE.

Species.	1904.			1905.			1906.			To 30th June, 1907.		
	Examined.	Infected.	Per Cent.	Examined.	Infected.	Per Cent.	Examined.	Infected.	Per Cent.	Examined.	Infected.	Per Cent.
Mus decumanus	19,689	309	1·55	13,283	87	·65	10,847	16	·14	9,254	13	·14
Mus rattus		63	·32		41	·24		32	·29		9	·09
Mus musculus ...	4,963	3	·06	3,497	1	·03	1,348	0	0	1,125	0	0

In the Ulmarra outbreak of the Clarence River district, New South Wales, investigated by Dr. R. T. Millard (*vide* Plague Report, N.S.W., 1905), rodents to the number of 2,683 were examined at Ulmarra for evidence of plague infection from 8th January to 10th March, 1905. "The second rat actually examined at Ulmarra was found to be plague-infected. This was on the 8th January. The last infected rat was found on 8th March. Plague infection was actually demonstrated in 106 rats and 3 mice. The infected rats were all of the *decumanus* species. This is not contrary to previous experience in this State, but is, perhaps, worthy of mention, in view of the theory recently suggested (in B.M. Journal, of 6/5/05), that *M. decumanus* is naturally immune to plague. It should, however, also be stated that of 1,128 rats examined at Ulmarra 1,125 were *decumanus* and 3 *rattus*. On the other hand, of 234 rats from Grafton (where there were no cases nor infected rodents) 172 were *decumanus* and 62 *rattus*" (*vide* Report, p. 20).

It may be mentioned in connection with the above statement that, at Ulmarra, the *rattus*, if more susceptible, might have succumbed first, and before mortality was observed among the *decumanus*, for mortality amongst rats had been intense long before the 8th January. "The report of previous rat-mortality was supported by the finding on 9th and 10th January of 50 rats and 65 mice mummified."

It is an interesting fact that, in all places where successive outbreaks have occurred, the *M. rattus* has been found more or less largely infected during the epizoötics observed.

On the other hand, specific instances can be cited to show that a more or less severe epizoötic can exist amongst the rats of the *M. decumanus* species without a resultant epidemic in human beings; or, at most, with only one or two cases in man.

In the Glasgow epidemic of 1900, it was noted that, notwithstanding careful bacteriological examination of large numbers of rats, no single instance of rat infection was discovered. "From the end of August till the middle of

November, 236 rats were caught within the plague area—mostly in the neighbourhood of infected houses—and no trace of the disease was discovered in any of them. . . the additional number of 124 rats was submitted for examination from all parts of the town during the winter, but in no instance was there any sign of plague." (Report on Cases of Plague at Glasgow, 1900.)

All the rats examined at Glasgow, and referred to above, were of the *decumanus* species. The outbreak was limited to 36 cases with 16 deaths between August and September, 1900, and there has been no recurrence of epidemic plague since that date.

In the Liverpool outbreak of 1901, there was a like absence of rat infection.

In Cardiff, however, plague made its appearance in 1901, among the rats (*M. decumanus*) on shore, but it was accompanied by only one human case.

During the outbreak of plague in Brisbane in 1904, of 88 rats found plague-infected on careful bacteriological examination, and discovered in one produce store in the South Brisbane area during the cleansing operations, all were of the *decumanus* species. No case occurred among the store employees.

At Childers, a small township near Bundaberg, in Queensland, a man died of septicæmic plague on 16th March, 1905. For some time previous to his death the rats were reported to be dying in hundreds within the township. So severe was the epizoötic that practically the whole rat-tribe of the town was decimated. Only one case in human beings, above referred to, occurred. In every instance the infected rats were found to be of the *decumanus* species.

In Section II. it will be shown that the plague-infected rat (*rattus*), however, is not the only factor concerned in the spread of epidemic plague. The agency (rat-flea) by which the infection may be conveyed from rat to rat, and rat to man, is of even greater importance in its *distribution* than the species of rats found within infected localities. The exact part played by the rat-flea has yet to be determined.

In Europe, including Great Britain, the usual flea found on *M. decumanus* is the *P. fasciatus*. The *P. cheopis* is extremely rare, and then only found on the rare *M. rattus*.

In India, the *P. cheopis* (Roths.) is by far the most common rat-flea.

In Queensland (Brisbane, Rockhampton, Townsville, and Cairns), in New South Wales (Sydney), and West Australia (Perth) the most prevalent rat-flea is the *Pulex cheopis*.

In Victoria (Melbourne), Tasmania, and New Zealand, the *P. cheopis* is the least prevalent; at Melbourne, the preponderating flea is the *Ctenopsyllus musculi*, and the same flea is found in the ascendant at Hobart in Tasmania.

In New Zealand, the *Ceratophyllus fasciatus* is said to be the flea most frequently found on rats.

Wherever epidemic plague has been observed, it has been remarked that its prevalence in any year of its occurrence has a marked seasonal feature. This fact, moreover, wherever fleas have been observed from the statistical point of view, finds its analogue in their numerical manifestations also. This has been noted in Queensland, in New South Wales, and is a common experience in India.

In dealing with the subject of fleas occurring upon the rats in Australia and in other countries (*vid.* p. 144-147), it will be shown that the distribution of the *Pulex cheopis*, both in time and place, has its counterpart in that of epidemic and probably epizoötic plague. Mention will also be made of the fact that *Pulex cheopis* is found on rats inhabiting the warmer portions of the earth, and a probable explanation of this phenomenon of geographical range of occurrence may be that the *Pulex cheopis* is susceptible to meteorological influences, which to an extent determine its distribution and seasonal prevalence.

In it we have—(1) a flea that occurs in connection with naturalised rats susceptible to plague throughout the world wherever plague epidemics are observed, and absent where they are absent; (2) a flea in which the plague bacillus may be harboured and in which it may multiply; (3) a flea that can convey plague to susceptible animals, and does so readily; (4) a flea that, included in the same group of these insects that contains the special Human Flea (*P. irritans*), feeds especially on the worst implicated domestic rat (*M. rattus*), and more especially on man than any other rat-flea; (5) a flea whose seasonal prevalence agrees to a very noteworthy degree with that of plague [*cf.* Sect. II., "Rat Parasites and Plague," p. 137, *et. seq.*]

The main fact then stands out that the *M. rattus* and the *P. cheopis* are closely associated with the spread of plague, and where one or both factors are absent the infection in epidemic form is not likely to occur independently of these agencies.

That the *M. rattus* is the true host of the *Pulex cheopis*, as the latter is the true intermediary host of the *B. pestis*, there is much reason to believe.

V.—CHRONIC PLAGUE IN RATS.

The question as to what becomes of the plague germ during the period of freedom, the season when neither plague cases nor acute plague amongst rats occurs, has often been asked: How is infection maintained through these intervals of apparent freedom? These recrudescences are particularly marked where epidemics acquire a very intense character. In India, especially, there are many instances remarked of recurrence of plague in the absence of any history of imported infection notwithstanding the discovery of this is made the object of special quest. Major S. Browning-Smith, I.M.S. ("Ind. Med. Gazette," XLI., No. 7, 1906), refers to this as implying latent infection. To explain it, he proposes a corollary to the Flea Hypothesis, which he sets forth in the form of possible factors determining the results—(1) Infective material absorbed by the larval flea and conveyed to a susceptible host; (2) reinvigoration of the plague bacillus that meanwhile has become non-pathogenic within the flea organisation, and prior to such conveyance; (3) the preservation of the plague bacillus from one season to another in the ovum or pupa stage of the flea.

Many epidemiologists have suggested that the interval between epizoötics is "bridged" by the persistence of rat-plague in a chronic form. This interval may be, and frequently is, of long duration; for, in the case of Sydney, as also of Brisbane, intervals of six to nine months were observed between some epidemics and epizoötics.

It is probable that under certain natural circumstances a reduction in the virulence of the plague organism is affected and a comparative immunity is conferred on rats. This immunity is real and capable of actual demonstration. The actual decrement of infectivity in rats cannot be ascribed either to lack of systematic observation during the plague-free months, or explained altogether by the seasonal breeding periods of the rat. This attenuation of virulence may be responsible for the conditions described by several observers as chronic rat-plague, and thus for the recrudescences alluded to.

This form of rat-infection, commonly, and probably erroneously, termed chronic plague, has been suggested by the fact that out of large numbers of rats caught at a time when neither human nor rat plague, as far as was known, existed, a very small proportion of the rats showed on *post-mortem* examination abscesses within the abdomen. From these abscesses organisms morphologically indistinguishable from *B. pestis* were obtained, and careful cultural and inoculation experiments also proved that these abscesses were due to plague bacilli. It has been further observed that the rats with these chronic plague lesions showed no other signs of ill-health.

The condition described as chronic plague in rats would appear to be different in the several accounts given by observers who have recorded it. Kolle described a chronic form of rat-plague manifesting itself in circumscribed caseous modules in the submaxillary and bronchial glands. Hunter observed that rats caught between epizoötics of acute plague suffered from chronic diarrhœa, and were emaciated in appearance. This observer also found that plague bacilli contained within areas of necrosed tissue consisting of cheesy material in the lymphatic glands and viscera were, although few in number, capable of causing acute plague in healthy rats.

In the September number, 1906 (Vol. 6, No. 4) of the "Journal of Hygiene," the Indian Advisory Committee give an account of the existence of chronic plague in rats in localities in India where plague is endemic. The conclusions arrived at by the committee were to the effect that the lesions were in every instance situated within the abdomen; that the animals killed furnished no indications that their health suffered in any way, and that of the animals inoculated directly from, and with subcultures of, the abscesses of rats with chronic plague only one survived.

In continuation of the investigation, the Committee describe (July, 1907, Vol. 7, No. 3, "Journal of Hygiene") forty-five cases of localised encapsuled abscesses containing virulent plague bacilli met with in the rats (*M. rattus*) in the Punjab.

In twenty-eight instances these abscesses were situated in the peripheral lymphatic glands, and in seventeen cases they were visceral.

These rats were in all cases trapped alive, and showed no other lesions nor signs of ill-health.

In Bombay, on the other hand, only one chronic plague rat was met with out of 17,000 plague-infected rats.

The report states that "it is not easy to see how these chronic abdominal abscesses can afford a source of origin for an acute epizoötic. The fact, however, remains that they are the only places where plague bacilli are known to occur in the intervals between the epidemics of acute rat and human plague in the Punjab."

In pursuance of our investigations in Brisbane we have found encysted abscesses in guinea-pigs recovering from plague after subcutaneous inoculation with *B. pestis*. In some of these abscesses virulent plague bacilli have been found.

Guinea-pigs convalescent from plague have been kept in the laboratory from one to two years, and then killed. One of these recovered animals was killed eight months after inoculation, and one after twelve months, but *post-mortem* examination did not reveal the presence of abscesses in either case. In the daily examination of rats at the Bacteriological Institute a certain proportion, about 2 to 5 per cent., of the rats was observed to have more or less pigment—the product of an inflammatory process—in and around what had been evidently an infected gland. These rats were all caught in Brisbane.

It was also found that, while rats at the beginning of, or during, an epizoötic were readily infected with plague, a certain proportion were found to be immune to inoculation with *B. pestis* towards the close of the epizoötic, and in the intervals of quiescence.

It was further ascertained that this acquired immunity lasted in many instances from six to twelve months.

On killing these rats (immune to plague), pigmentation of one or more lymphatic glands was found. This pigmentation was not general. As a rule it was confined to one gland series—cervical, axillary, or inguinal—but sometimes two or more were found "pigmented."

The pigment was undoubtedly the result of blood extravasation followed by destructive changes of the blood corpuscles.

During the year 1906, of 10,367 rats examined at Brisbane for evidence of plague infection, 228 rats, or 2·2 per cent., were found to have pigmented glands.

The following Report, by Mr. C. J. Pound, the Government Bacteriologist, is of great interest as bearing on this subject of immunity or insusceptibility of rats to plague:—

I.—OBSERVATIONS ON RATS WITH PIGMENTED GLANDS.

During the daily examination of large numbers of rats, it was observed that a certain number, approximately about 2 to 5 per cent., had a varying amount of pigment in and around the glands of the cervical axillary or femoral regions. From subsequent experiments there is evidence to show that this pigment is the result of blood extravasation—due to the hæmorrhagic condition of the glands in question—accompanying an attack of plague.

1. Rat (429) inoculated with virulent plague material from rat (465). Result: No signs of sickness. Killed two months later. All internal organs healthy. Pigmented condition of the femoral gland and surrounding cellular tissue on the opposite side to the seat of inoculation.

2. Rat (881) inoculated with virulent *B. pestis* from spleen of rat. Result: Died accidentally on 7th day. All organs normal, but there was a pigmented gland opposite to side of inoculation in the femoral region. No *B. pestis* detected.

3. Guinea-pig (888) inoculated with emulsion of pigmented gland of rat (881). Result: No symptoms of sickness, glandular enlargement, or rise of temperature.

4. Rat (890) inoculated with virulent plague culture. Result: No apparent signs of sickness. Died 19 days later from accidental causes. *Post mortem* showed all organs normal. Right inguinal gland only pigmented. No *B. pestis* detected. Control rat died of plague in 3 days.

5. Rat and three guinea-pigs inoculated with portions of pigmented glands of rat (890). Result: All animals manifested symptoms of sickness, with glandular enlargement. The rat died of plague in

Plate I.

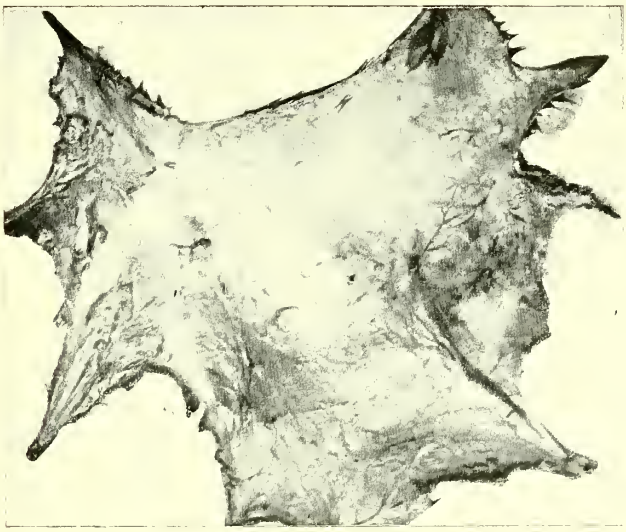


Fig. III.

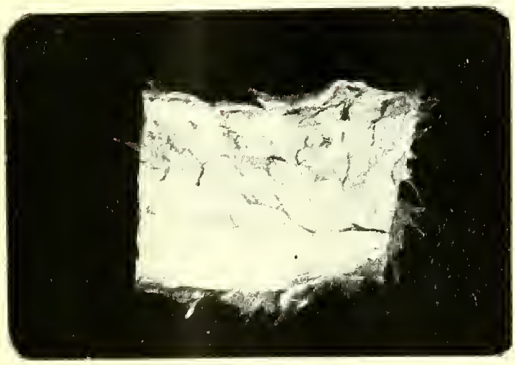


Fig. IV.

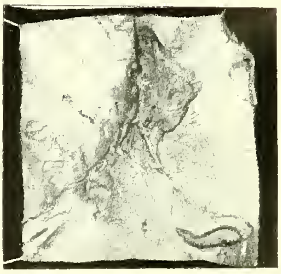


Fig. V.

C. J. Pound.

Photographs of portions of skin dissected from the inguinal region of rats recovered from plague, showing masses of black pigment in what were infected glands and also in the surrounding subcutaneous tissue.

14 days, with *B. pestis* in all organs. The guinea-pigs eventually recovered. The last-named guinea-pigs were, 14 days later, inoculated with virulent plague. Result: Two died in 5 days. The third died in 6 days. *B. pestis* were numerous in each case.

6. Guinea-pig (899) inoculated with pigmented gland from freshly caught rat. Result: Remained alive. No signs of sickness.

7. Two guinea-pigs (902-3) inoculated with pigmented glands from a rat. Result: Both remained alive, without showing any symptoms of illness. Thirteen days later the above guinea-pigs were inoculated with virulent *B. pestis*, when 902 died of plague in 3 days, and 903 remained unaffected.

8. Four guinea-pigs (912-5) each inoculated with a portion of pigmented gland from freshly caught rat. Result: 915 died of plague in 6 days. The other three became sick, but recovered.

9. The three guinea-pigs (912-4) were, 19 days later, inoculated with virulent *B. pestis* from culture. Result: (912) died of plague in 3 days (913) in 4 days, and (914) in 4½ days, with *B. pestis* numerous in all organs.

10. Rat inoculated with plague bacilli from rat. Result: Showed no signs of sickness. Seventeen days later reinoculated with plague from guinea-pig, but no signs of illness followed. Eighteen days later killed. *Post mortem* revealed a mass of pigmented glands in the inguinal regions, opposite to the seat of inoculation.

11. Rat inoculated with *B. pestis*. Result: No signs of sickness. Reinoculated 15 days later and died in 2 days. The *post mortem* revealed glands in the right inguinal region very much pigmented. All the organs normal. No *B. pestis* detected.

12. Guinea-pig (606) inoculated with blood from rat that had pigmented gland. Result: Died of plague in 3 days. This rat was undoubtedly plague infected, but presumably it had had a previous attack, as shown by the pigmented gland, which had not rendered it immune to the subsequent infection.

II.—ANIMALS AFFECTED WITH THE SUBACUTE FORM OF PLAGUE AFTER INOCULATION.

As a rule rats and guinea-pigs, the two most susceptible animals to plague, die from that disease in from two to four days after being infected either by feeding or inoculation. But, occasionally, in some individuals of these species, through being more resistant, the disease runs a much longer course, as is shown in the following specially selected cases.

1. Two rats (573-4) inoculated with virulent plague material from a recently naturally infected rat. Result: 573 died of plague in 3½ days; 574 showed no apparent illness until the 12th day, when it refused food, and gradually sickened, dying on the 20th day. The *post mortem* showed general infection. *B. pestis* numerous in all organs.

2. Rat (567) inoculated with spleen of naturally infected rat. Result: Symptoms of sickness appeared on the 4th day. Died of plague on the 10th day.

3. Guinea-pig inoculated with *B. pestis* from human case. Result: Appeared sick on the 4th day; continued to get weaker, and died on the 14th day. *Post mortem* revealed general infection. *B. pestis* numerous.

4. Rat (375P) inoculated with virulent culture of *B. pestis* from rat. Result: Appeared sick for three days, but completely recovered. Ten days later reinoculated with spleen from plague guinea-pig, and died of plague in 2 days.

5. Rat (89) inoculated with virulent plague culture. Result: Sick for a few days, then appeared to recover, but eventually died in 19 days.

6. Rat (590) inoculated with virulent plague spleen from guinea-pig. Result: Died in 15 days. Internal organs almost normal. Few *B. pestis* in spleen.

7. One rat (*Mus decumanus*) and a black rat (*Mus rattus*) were each inoculated with spleen pulp from plague guinea-pig. Result: No symptoms of illness whatever. These two rats were repeatedly fed and inoculated with virulent plague material for over two years, but remained unaffected.

8. Rats (573-4) fed with liver of plague rat. Result: One died of plague in 3½ days, and the other died on the 20th day. *Post mortem* showed general infection. *B. pestis* numerous in both cases.

III.—IMMUNITY.

1. A guinea-pig was inoculated with virulent *B. pestis*, and 24 hours later injected with 5 c.c. Yersin curative serum. Result: Became obviously ill for about a week, with enlarged gland and high temperature, during which time it lost weight considerably. Then it gradually improved and remained in good health. Two years and two months later it was inoculated with virulent *B. pestis* without showing any signs of sickness. A year later it was again injected with virulent *B. pestis*, but without producing any noticeable effect. This guinea-pig eventually died from accidental causes some eight months afterwards. Each time this animal was inoculated a control guinea-pig was also inoculated, every one of which died of plague within 5 days.

2. A guinea-pig injected intraperitoneally with 10 c.c. of Yersin curative serum, and 24 hours later inoculated inside the thigh with virulent *B. pestis* from a rat. Result: Became very sick and emaciated, suppurating gland at point of inoculation; had a high temperature for three weeks, then gradually recovered. Ten months later this animal was again inoculated with virulent *B. pestis* without producing any signs of sickness.

3. A very old guinea-pig, inoculated with material from a human case. Result: Showed pronounced symptoms of plague for about a week, then recovered. Fourteen days later reinoculated without showing any signs of sickness. Ten months later this animal died from old age.

4. Guinea-pig (367) and rat (368) were each inoculated with lymph from the femoral gland of a plague patient affected with a mild form of the disease. Both animals became sick, and remained so for a few days, but ultimately recovered. On the 14th day each animal was reinoculated with spleen pulp from a plague guinea-pig (380). Result: The rat died in 3 days with general plague infection. The guinea-pig remained alive, having shown no signs of sickness from the second inoculation.

IV.—DIRECT INOCULATION OF PLAGUE BACILLI FROM RAT TO RAT.

Many observers have demonstrated that plague bacilli cannot be transmitted from rat to rat beyond the third remove from the original culture or naturally infected animal. This result is considered by some to be brought about by the attenuation of the organism, and by others to the frequent immunity that exists among rats in the locality where the animals used for experimental purposes are caught.

For the purpose of confirming or disproving these observations, the inoculation of plague from rat to rat was attempted in a series of eight separate experiments. In each of these experiments a start was made from a naturally infected plague rat found in some part of the city—principally produce stores—where previously many infected rats had been caught.

The inoculations were all subcutaneous in the left inguinal region, and with spleen material only; the quantity about what could be picked up in the small loop of a platinum needle. Further, it may be of interest to know, the experiments were conducted at a time when plague was epizoötic among the rats. In every case the animal was kept under observation ten days before it was inoculated.

SERIES A.

No. 1. Species M.D. Died of plague in 3 days. *B. pestis* numerous.
,, 2. ,, M.D. ,, 5 ,, *B. pestis* numerous.
,, 3. ,, M.D. ,, 15 ,, *B. pestis* few.
,, 4. ,, M.D. ,, 4 ,, *B. pestis* numerous.
,, 5. ,, M.D. ,, 5½ ,, *B. pestis* numerous.
,, 6. ,, M.D. ,, 2½ ,, *B. pestis* numerous.
,, 7. ,, M.D. Remained unaffected.

SERIES B.

No. 1. Species M.D. Died of plague in 3 days. *B.pestis* numerous.
,, 2. ,, M.R. Killed on the 19th day. No *B. pestis* detected. Pigmented glands in axillary and femoral regions. A guinea-pig and a mouse inoculated with portions of these glands remained unaffected.

SERIES C.

No. 1. Species M.D. Died of plague in 2½ days. *B. pestis* numerous.
,, 2. ,, M.D. ,, 2 ,, *B. pestis* numerous.
,, 3. ,, M.D. ,, 7½ ,, *B. pestis* numerous.
,, 4. ,, M.D. ,, 11 ,, Spleen, liver, and lungs normal. No *B. pestis* detected. Both inguinal glands and tissues were pigmented. A guinea-pig inoculated with portions of these pigmented glands remained unaffected.

SERIES D.

A *Mus decumanus* rat, a guinea-pig, and a mouse were inoculated. The rat remained alive, while the guinea-pig and the mouse died of plague on the 4th and the 2nd day respectively.

SERIES E.

No. 1. Species M.D. Died of plague in 5½ days. *B. pestis* numerous.
,, 2. ,, M.D. ,, 3½ ,, *B. pestis* numerous.
,, 3. ,, M.A.R. ,, 12 ,, *B. pestis* numerous.
,, 4. ,, M.D. Remained unaffected. Nineteen days later reinoculated with virulent plague, but failed to exhibit any signs of sickness. When this rat was killed two months later it was observed that the femoral glands were pigmented, but a guinea-pig inoculated from these glands remained unaffected.

SERIES F.

A rat (M.D.) inoculated remained unaffected. Sixteen days later reinoculated with spleen from plague rat in Series A. Result: Remained unaffected. Killed 14 days later. No appearance of plague or pigmented gland, and no *B. pestis* detected.

SERIES G.

A rat (M.D.) was inoculated. Result: Remained unaffected. Reinoculated 14 days later from plague guinea-pig. Result: Remained unaffected.

SERIES H.

No. 1. Species M.D. Died of plague in 3½ days.
,, 2. ,, M.D. Remained unaffected.

The result of these experiments would appear to indicate that, in the epizoötic area, there must be a number of rats that are either naturally immune or have acquired immunity after suffering from a localised form of glandular plague infection.

It will be noted that, where the passage of the bacilli was carried on through six removes, although the second animal did not die until the 15th day, the last one died of acute plague in 2½ days, showing that there had been no apparent alteration in the virulence of the organisms.

In the Indian Commission Report (1906), it is stated that twenty-six passages from rat to rat were effected by subcutaneous inoculation of plague bacilli without having recourse on any occasion to cultures isolated from rats. Moreover, the virulence of the bacillus used did not undergo any alteration. In the second passage only one rat was inoculated. In eighteen of the series three rats were used, and, in seven instances, six rats were inoculated with the same material.

This method naturally increased the chances of recovering the bacilli in sufficient numbers to ensure the continuation of the passages.

It is somewhat interesting to note that, in the fifth passage, one of the three rats inoculated did not die of plague, while in the sixth, eleventh, sixteenth, and twenty-second passages only one of the three died of plague.

On seven occasions six rats were inoculated at one time, although the results are only given of three rats in each experiment. It is, therefore, obvious that it would have been possible for the experiment to have terminated much earlier than it did, had only one rat been used for each passage.

Section II.

RAT PARASITES (FLEAS) AND PLAGUE.

VI.—INTRODUCTION.

It is in accordance with views expressed in this and in other Reports on Plague—

(1) That the disease as it occurs in man, and in rats, amongst other mammalia, may be considered as one; and that the facts, connected with plague epidemics and plague epizoötics, imply a causal relation between one and the other;

(2) That with regard to the more common type of plague (*i.e.*, the bubonic form) data connected with its epidemiology, symptomology, and pathology point to its origination in inoculation;

(3) That this inoculation is effected by an external parasite consorting both with rats and man and passing on occasion from one to the other; and

(4) That a flea best fulfils these conditions. This is the Flea Theory of the origin of the Bubonic plague in plague epidemics.

It becomes a matter, then, of importance to consider, in connection with any Report on Plague, the subject of Fleas and the nature of their association with Rats, a subject that may be conveniently dealt with under several separate headings, as has been attempted in the succeeding portion of this Report.

This section of the Report reflects to a considerable extent the special work of Mr. H. Tryon, the Government Entomologist of the State, whose thorough attention to the subject of Rat Fleas was shown already in 1902 by an account (unpublished) of the "Pulicidæ of the Naturalised Rats of Queensland,"[*] based on material furnished by me. His carefully compiled contribution deals with the following technical topics:—The Life History and Habits of Fleas; the Rat Fleas of the world as well as those of Australia, and their distribution; the Rat Fleas of special Rat species, and the distinguishing features of those met with in Queensland, all matters of great importance in dealing with the Flea Theory of Plague in connection with the consideration of its ætiology.

VII.—LIFE HISTORY OF FLEAS AND THEIR HABITS.

[*Vide* PLATE II.]

The life history of the commoner rat flea (*Pulex cheopis*) has apparently not been recorded. There is, however, considerable uniformity in those of other living Pulicidæ. The following description pertaining to the subject may therefore serve as a guide to the understanding of what occurs in the insect in question:—

Egg Stage [Plate II., Figs. 1 and 1A.]—The flea is born in the egg form. These eggs are small and oval in outline; those of ***Pulex irritans*** being 0·7 mm. long by 0·4 mm. broad (Railliet), those of ***Pulex serraticeps*** about $\frac{1}{16}$ inch or 1·5 mm. (Verrall.) In colour they are whitish with a porcelainous surface texture, a remark that especially applies to the dog and cat flea, but some species have brown eggs (Theobald). A remarkable feature connected with them is their very thick shells, of two layers, the inner of which is composed of strong epithelial cells, whilst the outer is muscular. Dr. L. Landois, in his "Anatomie des Hundeflohes," has given a detailed description of their special structure. [*Cf.* Fig. 1A, Plate II. *a.-d.*, Outer or muscular layer; *b*, Muscles; *c*, Epithelial or cellular layer; *d*, Egg contents]. When one treads upon flea eggs reposing on a smooth hard surface, the strength of their shells is suggested by the resistance which they offer to being broken, and the sound that accompanies this act. This peculiarity characterising them suggests also that the flea may, if necessary, persist long in the egg stage, and that during this time it may tolerate considerable changes in its environment. Railliet states that each female of *Pulex irritans* deposits eight to ten eggs. Theobald, in writing of fleas generally, adds "at a time." In *P. serraticeps*, and the same remark apparently applies to all fleas, the total number laid is far in excess of this. Landois, in the work above, especially refers to this, and states that in the dog flea there are five ovisacs on each side of the body, and in one of these may be discerned from seventy-five to eighty eggs in various stages of development, which gives a possible production of 800 from a single insect. Anyone who has examined the spot where a flea-infested animal has lain can appreciate this prolificness. This remark suggests the further one, that the ordinary fleas do not glue them on to the hair, or insert them beneath the skin, as is the case with other ecto-parasites, but lay them on the fur to which they are but loosely attached, and whence they are readily shed. Referring to *P. serraticeps*, Verrall writes: "As the cat walks or runs about, the eggs are being constantly scattered round, often in great numbers. On one occasion I was able to collect fully a teaspoonful of these eggs from the dress of a lady in whose lap a half-grown kitten had been held for a short time." These eggs may occur on any object whereon an animal harbouring fleas may even temporarily rest. Moreover, when deposited—as with ordinary dust—they will soon settle in any interstices that these objects may exhibit. Left on a wooden floor, for example, the wind or ordinary brushing will usually transfer them to the cracks separating the individual boards, and in the fine *débris* that accumulates in these places they may find both shelter and aliment. This remark applies to the more common domestic fleas, but there are no grounds for concluding that it does not hold good with respect to the special kinds associated with rats. Jules Kunckel, in his "Observations Sur Les Puces," expresses the opinion that egg-laying on the part of fleas is especially favoured by circumstances that compel their hosts to remain in one place, especially dwelling upon the periods of hibernation, the care of young, and senility with its attendant infirmities, and he might have mentioned sickness.

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[*] It may be of interest to point out that at this early date Mr. Tryon already recognised that the most prevalent Rat Flea of Queensland was an undescribed species (afterwards named by Rothschild, *Pulex cheopis*). "In using the binomial *Pulex pallidus*," wrote this entomologist, "for what is evidently our common Rat Flea, and the one often exclusively present on Rats, I have written the authority for this name Tidswell, *and not* Taschenberg. By this it is implied that the flea referred is one that Dr. Tidswell, in his reports, mentions and figures under *Pulex pallidus*, and that is not the *pallidus* of the European specialist."

The eggs of the human flea (*Pulex irritans*) contain fully-formed larvæ after six days during the summer months; and in the winter time, if kept under warm conditions, do so when twelve have elapsed (Babiani). Verrall assigns two weeks for the hatching of the eggs of *Pulex serraticeps*. From the observations of Pergande, quoted by L. O. Howard and C. L. Marlatt (Principal Household Insects), this period for the same fleas' eggs in the latitude of Washington is usually from 2-4 days' duration only. The length of the time during which hatching may be suspended, should the eggs experience cold, has not been ascertained.

Larval Stage.—[Figs. 2, 2*a-b*.] The larva is a minute worm-like object, and, to enable it to rupture the shell and so effect its exit from the egg in which it has developed, is endowed with a small depressed yellowish-brown horn on the summit of its head, that has been both figured and described by Jules Kunckel; his observations relating to this occurrence in connection with the young of *Pulex felis*, and as manifested by *P. fasciatus* also. This disappears early on the insect's molting.

These young fleas are narrow, elongated, greyish-white, footless, maggot-like insects, having almost transparent bodies. They are composed of twelve distinct oblong segments in addition to the head, each of which bears behind a bunch of long hairs, the terminal one possessing also, in addition to other hairs, two prominent curved projections. This larva, when newly hatched, is about $\frac{1}{16}$ inch long. It is capable of a wriggling forward movement, in which the appendages, the body hairs, and certain hooks on the under surface of the head assist. Respiration is provided by rows of spiracles (*s*), one to each segment, on either side of the body. The mouth-organs [Fig. 2*b*] are structurally distinct from those of the adult, and are—unlike them—not adapted for sucking purposes. They comprise amongst other parts two curved toothed mandibles (*Md.*), each terminating in a sharp point, and two maxillæ (*Mx.*), each having two plate-like divisions with circular cutting toothed free borders.

These flea-larvæ live in the crannies wherein they have been born—yielded by the soil beneath houses, floors, carpets, boards, old bags, &c. (In the case of the *Pulex irritans*, Theobald has reported its occurrence also on man's body, but this must be quite exceptional.) They occur in association with the dust and *débris* of various origin that has accumulated in these spots, but, owing to their poor powers of locomotion, although endowed with considerable wriggling activity, do not, it is considered, travel far. Usually more or less material of a fluffy nature, derived from their surroundings, adheres to their bodies, and so serves to mask their proper appearance.

Plate II.

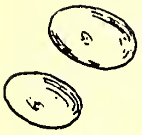

Fig 1.

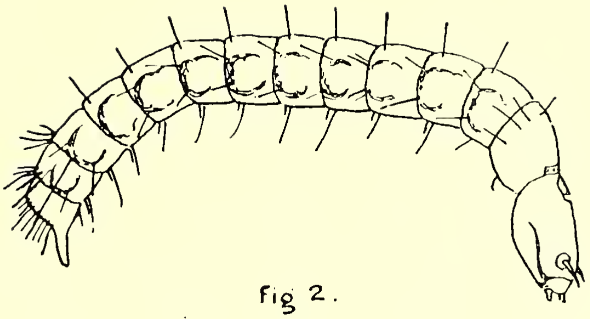

Fig 2.

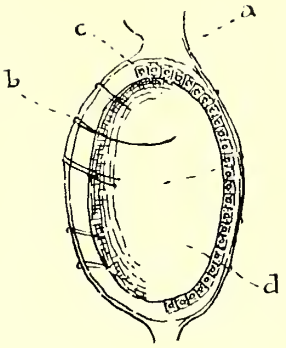

Fig 1a.

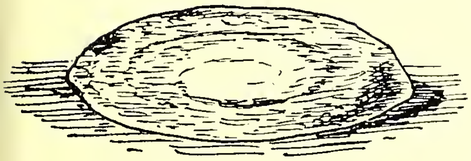

Fig 3.

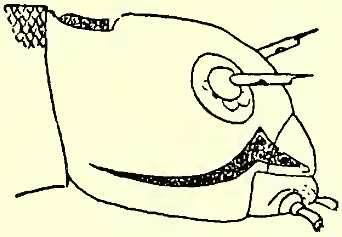

Fig 2a.

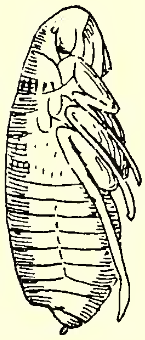

Fig 4.

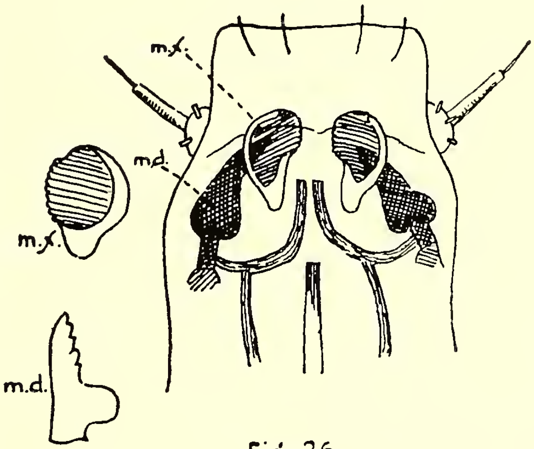

Fig 26.

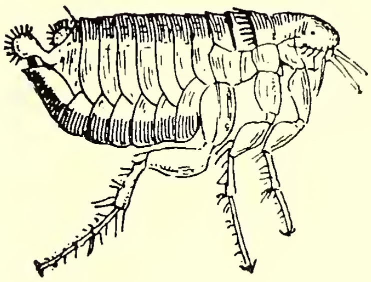

Fig 5.

LIFE-HISTORY OF FLEAS.

Their mouth organs—as Kunckel has remarked—being adapted, on the one part, for rending and tearing, on the other for sawing and cutting, suggest that they feed on various organic substances: they have been reared on dead flies (Leewenhoek), on the fresh blood of pigeons, on the bodies of dead mosquitoes, and on dry and pulverised blood (Roesel).

The observation that their stomachs, as viewed through their transparent bodies, were often red, and, therefore, contained blood, formerly led to the adult fleas being accredited with remarkable maternal instincts, it being suggested that the mother insects nourished their young by degorging into the mouths of the larvæ blood that they had reserved for this purpose, after the manner of ants and bees. Defranc, however, in 1824, observed that amongst the eggs of fleas there occurred dark-coloured particles almost as rounded as themselves, on which the larval fleas evidently fed. These particles, he found, were of the nature of dried blood, and he was led to conclude that they emanated from the bodies of the parent flea through artificially produced fissures ("Il suppose que les femelles ont le soin d'ouvre la peau et de laisser couler le sang pendant un certain temps et que ce sang coagulé produit les grains en question—Kunckel"). However, the entomologist Kunckel, to whom I am indebted for this information, discovered that these particles were really nothing but flea-excrement.

When flea-infested dogs or cats are leaving spots where they may leave eggs of their parasites, these dark particles, resembling dried congealed blood, are frequently to be observed. They may be flea-excrement, as suggested by Kunckel, but more likely the droplets—dried—of red blood that fleas emit from their extremities from time to time when engaged in absorbing the life-juices of their hosts.

E. A. Butler, in writing of the common flea (Knowledge, 1st January, 1890), after referring to the fact that "the alimentary canal, when gorged with blood, can be rapidly emptied by the insect and its contents ejected with considerable force when a new and good supply of food presents itself before the last meal is disposed of," adds "The dark stains on the linen, that indicate where fleas have been, consist of their dried excrement, and are composed of the undigested remains of the blood corpuscles contained in the food."

[Feeding on these substances, it may be assumed that an entry is thereby gained into the flea-system of whatever bacteria may be associated with them.]

Writers agree generally that during warm weather the flea-larva rapidly grows, and that under this circumstance they mature in about twelve days. Under other conditions, as Pergande has noted, larval existence in the cat-flea may be as short as eight and may extend to twenty-four days. This is stated both with regard to the common flea (*Pulex irritans*) and the dog and cat flea (*P. serraticeps*). During this period the alterations affected are these of size only, and are accompanied by several ecdyses, or moltings, of the skin.

H. Osborn, referring to the former of the above fleas, states that on the eleventh day the larva discharges the contents of its alimentary canal becoming uniformly pale.

The Pupa Stage.—[Figs. 3 and 4.] Soon after becoming full-grown, the flea-larva forms a chamber as it were in the dust and *débris* that surrounds it, and therein spins a small whitish, very delicate, dust-covered cocoon, usually affixed by one side, within which it changes to a pupa. [Fig. 3.]

This is only if genial weather occurs. For, as writes F. V. Theobald, "All the larvæ do not pupate at the same time. Some live through the winter, and hatch in the spring. (An account of British Flies, p. 23—1892.)

In some cases flea-larvæ may transform to pupæ without first forming a cocoon (Roesel).

The pupa [Fig. 4] is unlike the larva in appearance, being much stouter and of smaller dimensions, and, in fact, more nearly resembles the perfect insect. Thus we can discern the well-developed limbs of the latter, but as yet folded up and inactive. Moreover, the colour is more pronounced, yellowish instead of transparent white, much darker when no cocoon is formed, and also gradually acquiring a deeper hue with age, being ultimately brown. Dr. O. Taschenberg quotes Ritsema to the effect that already the males and females may be distinguished by outward features. Thus in the male pupa, the back is concave and the hind body terminates in two projections, whereas in the female the back is convex and terminates in a single one. (Of course, the pupal is an inactive stage, and whilst it lasts no food is or can be partaken of.)

Railliet, writing of the ordinary domestic flea (*P. irritans*) and referring to summer weather, states that about twelve days are spent by the flea in the pupa condition; and a similar statement has been made by other writers regarding its duration in the ordinary dog-flea. Pergande, however, conducting his observations during the summer months in the latitude of Washington, found this in the few instances that came under his notice to vary from five to seven days only.

(This would indicate that, regarding the life of the flea as a whole, consisting of the egg, the larval, the pupal, and the adult phases, from four to six weeks are occupied with it, this time varying in extent as the weather is warm or moderately cold; to this period must be added, again, the time occupied by the imago, or perfect insect, in arriving at sexual maturity and depositing its ova.)

It has been already stated that during winter months the duration of any phase in the life-history of the flea may be prolonged, the ordinary metamorphoses being temporarily suspended.

Seasonal Occurrence.—From the foregoing statements it may be concluded that in certain localities there may be definite periods separated by intervals of about six weeks' duration, during which fleas are prevalent, and that during the winter months in temperate regions the numbers of adult fleas, or of fleas at large, may be reduced to quite small proportions. The latter remark, however, has probably exclusive reference to extra-tropical occurrences.

One well-acquainted with the habits of the insects of Great Britain has informed us that in the south of England there are, generally speaking, three periods of the year during which fleas are prevalent, separated by intervals of remission from their attacks:—(1) About the end of March; (2) the middle of June; and (3) towards the end of August or early in September. He spoke from memory only. Also that so noteworthy was this, that, in the county of Kent, the fact had been commemorated in a well-known popular saying, "Oat-sowing, oat-blowing (blossoming), and oat-mowing," descriptive of the time of such occurrences.

The English Entomologist (S. A. Butler), previously referred to, writes on the subject, "In folk-lore, the 1st of March is intimately associated with fleas. It is still a practice in Kent to keep the doors shut on that day to prevent the fleas from entering, and in Sussex the door steps are swept on the same day for the same purpose, and thus it is believed that immunity from their attacks will be secured for a twelvemonth."

Observations regarding flea prevalence in Southern Queensland have not been sufficiently systematised to definitely indicate a periodicity of occurrence of this nature, but that it obtains is almost certain. A correspondent, referring to Brisbane experiences, stated that in one year there occurred " a very early spring," and that thus fleas were already prevalent in August, and that in another year (1906-7) they were locally terribly prevalent about Christmas time.

Another correspondent in referring to flea-prevalence, at the Southern border of Queensland, stated that they were exceedingly numerous in the light sandy soil adjoining his tent, frequenting it also. This was early in October. They were again very numerous at a short distance to the south during the latter part of November. It has been observed that fleas were very numerous in the Brisbane district at times during the period January to March, a noteworthy instance of flea-prevalence occurring at the end of the latter month. An observer, however, again encountered them in large numbers at Cape Moreton, on 16th-17th June, within a dwelling, and, referring to a Rockhampton occurrence, a witness deposed that they occurred in the precincts of certain stables during our midwinter also.

In Reports on the outbreaks of plague at Sydney, the prevalence of fleas in numbers in dwellings are incidentally mentioned in connection with the dates 8th January and early in March, and during the month of June. Further, the author of these, Dr. J. Ashburton Thompson, writing in October, 1906, on the "Epidemiology of Plague," states: "It (*i.e.*, March, April, and May) is also the season of fleas. From about February onwards is the time when sandy patches, far removed from dwellings, begin to swarm with them; when domestic dogs are worried by the species that infests them as at no other time of the year; and when one or other of the rooms in many a house which is neither dark nor dirty, but often of the better class, becomes for a time overrun with them. . . . but . . . these insects are to be found all the year round, and in numbers in the warm, dark places where they best breed."—("Journal of Hygiene," VI., 564-5.)

Observations regarding the species of *Pulex* figuring in these seasonal infestations indicate that this is in the majority of instances the dog and cat flea, *Pulex serraticeps.* They may, however, be fleas that are addicted to attacking man. Writing of a Sydney occurrence in March, 1900, it is stated "the bodies of the younger children were almost literally covered with their punctures."—(J. Ashburton Thompson: "Report on Outbreak of Plague," 1900, p. 25.)

Circumstances, however, may bring it about that fleas may appear excessively numerous at a time subsequent to that at which their numerical increase has taken place. Adult fleas are endowed with persistent vitality under untoward conditions of life, and thus can undergo long fasting. So, when a host has not been available to yield them their proper food, they will assert themselves when one is forthcoming many days after their first appearance on emerging from flea cocoons. Ordinarily speaking, too, fleas may under some circumstances manifest, relatively speaking, considerable longevity, remaining inactive between successive feedings, during which their transport from place to place may be effected. And it must be remembered in this connection that the ordinary Pulicidæ are not by any means continuously-abiding animal parasites.

This fact, and a consideration of the manner in which the eggs of fleas become scattered about, will suggest, what is so common an experience, that fleas will not only be met with at large, but that they may habitually occur generally all around the spots where their proper hosts exist or are domiciled.

The adult insects are, however, very susceptible to climate changes, a remark also that applies to the larvæ, and is indicated by the fact that their rearing to maturity is somewhat difficult. These fleas invariably seek shelter in rainy weather. This habit, then, has also to be taken into consideration in accounting for their prevalence (by which is generally intended prevalence in domiciles, where the annoyance their bites occasion leads to their presence being recognised) at any particular period of the year.

Even during winter in Brisbane districts—for example, in June, when it is thought fleas are less numerous than at other periods of the year—the advent of rain may cause the few occurring in the immediate vicinity of houses to enter them and to annoy the human occupants, who are wont even then to speak of "bad nights for fleas."

Darwin ("Voyage of the 'Beagle,'" page 344) when exploring Coquimbo had the following peculiar experience. He states: "I enjoyed my nights' rest here; from a reason which will not be fully appreciated in England—namely, the absence of fleas. The rooms in Coquimbo swarm with them, but they will not live here (in the mountains overlooking that city) at the height only of 3,000 feet or 4,000 feet." This, Darwin concludes, was due to some other cause (not ascertained) than diminution of temperature arising from an elevated situation. It indicates that complicated circumstances may possibly influence the degree in which fleas are prevalent.

Special Conditions Influencing their Occurrence on their Hosts.—Apparently the association or consortism of fleas with rats may be of a special character from the point of view of the occurrence in time. Thus Brisbane experiences point to the fact that, during the occurrence of an epizoötic, fleas are more numerous on rats—both those that are plague-infected and those that are plague-free—than when this is absent.

This is agreeable to the experience elsewhere. Thus we find Dr. Ashburton Thompson referring to the investigations of the Microbiologist of the Board of Health, New South Wales, stating as follows:—"Dr. Tidswell has been struck in this connection with the fact that, during the epizoötic, fleas were abundant on all the rats brought to the laboratory, while, as soon as evidence of its continuance had failed, fleas became so uncommon on the rats, which were still brought in large numbers, as to be quite insufficient for purposes of laboratory collection (see "Ectoparasites of the Rat," page 71). The abundance of fleas on rats during continuance of the epizoötic was not specially associated with illness of the animals;

it was generally observed on those that were perfectly healthy—as well as on others."—("Report on Plague Outbreak," Sydney, 1902, page 64.)

Dr. E. H. Hankin, writing on the "Epidemiology of Plague," relates to somewhat similar experience with regard to fleas infesting domestic cats :—"In the spring of 1901 (he states) I was studying fleas obtained from cats and dogs in Agra, and found no difficulty in obtaining as many of these insects as required, but at the commencement of the hot weather I found that the fleas on the cats living in my laboratory had suddenly and completely vanished. I offered half a day's wages to my servants for every flea they could catch. Not a single flea could they produce. A restricted plague epidemic that had been going on at Agra suddenly came to an end (he adds) at the time of the disappearance of the fleas."—("Journal of Hygiene," Vol. 5, January, 1905, p. 80.)

Situations in which Fleas at large occur.—With regard to the situations in which fleas occur at large, the fact alone that they originate from eggs that fall or are shed from the bodies of the animals that serve them as hosts will have suggested that they will be met with wherever those animals sojourn, temporarily repose, or happen to succumb. Accordingly, the fleas of domestic animals, such as cats and dogs, will occur in, or within, the precincts of houses, sheds, &c., but not necessarily very close thereto. This latter remark also applies to the rat-fleas proper, and especially to those in association with the more domiciliary *Mus rattus*, and its variety *M. Alexandrinus*, and to a somewhat less degree to the fleas of *Mus decumanus.*

How this is Influenced by Meteorological Conditions.—Moreover, fleas habitually shun the light, and permanent moist conditions are usually inimical to them, at least when they are in the larval condition. Accordingly, when occurring in the open it is where the soil is dry and sufficiently loose to admit of their entering and so ensconcing themselves—whether it be sand, dust, or ashes. The partiality on their part to darkness was incidentally demonstrated in the course of an experiment conducted by the Indian Advisory Committee. Two small rooms were provided to which fleas from rats has equal access, one, however, differing from the other by having a roof-light. The dark room yielded 224 of these insects, and the illuminated one 54 only ("Jour. Hyg." V. 453). Their most favoured haunts are floors, the ground beneath these, and other shaded spots, thickly strewn with dust, dirt, or vegetable *débris*, little exposed to disturbance. In such situations they may occur in immense numbers. "I have seen (writes W. W. Froggatt) dry stack sweepings taken out of pigsties in the country (New South Wales) so thick with fleas that the stuff was alive." Places that afford them harbour and concealment may be again objects occurring in their haunts—*e.g.*, clothing, bags, bales, skins, old fodder, &c.—and dwelling within these, they may be transported over long distances.

Moreover, they are intolerant of high temperatures, and it is probably the experience that sunshine and heat serve to some extent to banish them from domestic animals that has led cats that are nocturnal in their general habits, to favour a concurrence of the two, lying on the open to enjoy their insectifugal influence, just as one of ourselves might hang out his blankets under the same circumstances to rid them of the domestic insects in question.

E. H. Hankin has, as already stated, remarked (referring to observations at Agra) the disappearance of fleas from dogs and cats at the commencement of the hot weather ("Jour. Hygiene," Vol. 5, p. 80, 1905). Although, when harboured by a fur-bearing animal of semi-aquatic habits (*e.g.*, certain rats), the adult flea is apparently little affected when such animal resorts to water, finding, no doubt, meanwhile sufficient air imprisoned in its coat for respiration; it, when at large and away from its host, shuns moisture and wet. Thus, with the advent of rain, fleas migrate to the shelter afforded them by houses, sheds, and other structures (a most noticeable event after a "spell" of dry weather), and thus a high degree of infestation may be remarked, suggestive of a greater flea prevalence generally speaking than really obtains. This has to be taken account of in considering the seasonal occurrences of Pulicidæ.

Fasting Habits and Accommodation to different Hosts.—There are grounds for concluding that when fleas are living apart from the animals on whose blood they subsist, either owing to their not having had an opportunity, subsequent to their issuing from the flea-pupa, of attaching themselves to these, or owing to their hosts having died, they will, if other conditions favour their existence, exist a considerable time without food. This statement may apply to the larvæ also, for on examining the Pulicidæ present in rooms, that have remained closed for many days, and that have been found to be grossly flea-infested, the fleas present have generally been male individuals of diminutive growth, a state of things suggestive at least of earlier inanition. This capacity for fasting renders fleas all the more voracious when an animal that will yield them their sanguineous diet comes upon the scene, and not only so it will result in a flea proper to one host, or especially partial to it, attacking another to which usually it is not parasitically related. This is a very common experience so far as concerns the dog and cat flea (*P. serraticeps*) and man, that when either of the animals whence it derives its popular designation is available rarely only annoys him by its bite. The same has been found to occur in the case of the rat-flea (*Pulex cheopis*).

Thus Hankin referring to the observations of Captain Liston (I.M.S.) has stated: "He found that man rarely harboured the rat-flea under normal conditions. Out of 246 fleas caught on man, in the absence of plague, only one was a rat-flea. But, during a plague outbreak in a lodging-house, out of thirty fleas caught on the human inhabitants not less than fourteen were rat-fleas."—("Jour. of Hygiene," 1904, p. 83.)

The latter on *a priori* grounds, however, would appear to be a more probable event, since it more closely approaches in its structure the human flea (*P. irritans*) than does the one affecting our animal companions. This accommodation to different host, under exceptional circumstances that might have been illustrated by reference to the habits of other flea-species, has a far-reaching significance when fleas are serving in the rôle of intermediaries for the communication of specific diseases.

Their Occurrence on Their Hosts.—The relation between these ecto-parasites and their hosts has another interesting feature—this is, when fleas abide on the bodies of animals, they frequently favour one part more than another. This probably is less pronounced than is the same feature exhibited by external parasites of other groups; but still it is noteworthy. Thus with regard to rats, fleas more commonly occur, when infesting those rodents, about the head and neck than elsewhere; and a study of the mode of occurrence of cat-fleas on their namesake suggests that the frontal and temporal regions of this animal are especially affected. Whether any such partiality is displayed by fleas in the case of man has not been noticed, but the matter has been complicated by the fact that he is habitually clad. In his case, however, fleas rarely resort to the hair of the head; sed de pudendi indumento aliter.

In the foregoing remarks, allusion, of course, is made to the spots where fleas congregate for feeding purposes. On first alighting on their host, or when moving over the body of same, they may be encountered in any part thereof.

Their Feeding Habits—General Features.—Before dealing with the method itself of their feeding, attention may be called to the fact that fleas, unlike ticks (Ixodidæ) and some other external parasites, do not, as a rule, complete their meal with the blood derived from one spot on the body of their host only, and thus may inflict several bites before this procedure has been completed. This habit has been well described by C. F. Baker in the following words :—"Most fleas are epicures in their blood-sucking habits, where mosquitoes are gluttons. They do not settle and gorge themselves as do the mosquitoes, but pass rapidly from place to place and bite often. A single flea has been observed to bite so as to leave a dozen or more inflamed spots in as many minutes, and yet its abdomen showed no extraordinary dilatation."—(*Science*, 15th September, 1905, p. 340.)

It is again noteworthy that a flea, on having satisfied its appetite for the time being, will remain inactive in some retired spot for hours together, in order probably to digest the food it has partaken of, and in some instances lies ready, after such interval has elapsed, to resume its feeding habit. And, indeed, it has been held by some that it is only met with on the body of the animal that affords it sustenance when this animal has moved off, whilst the flea is still occupied in feeding. This is a feature in its economy that is conducive to its passive conveyance from place to place, in connection with one article or another, without detriment resulting from any enforced fast that this may necessitate.

Persistence, or the Reverse, on Hosts.—Fleas are much addicted to infesting sick and infirm hosts. This, in the case of occurrences of fleas in connection with rats, may be accounted for by the fact that these hosts may, under these circumstances and in the face of other ills, be indifferent to their presence, or be physically unable to resort to efforts for ridding themselves of them. It is considered, however, that this is not the sole explanation of this phenomenon.

Fleas that have once taken up their abode on the body of their proper host (*e.g.*, the cat-flea on the cat) apparently seldom leave them while such animal remains healthy and exists under normal conditions; but should the animal on which they occur be only, so to speak, a facultative host, their sojourn on it is temporary and transitory only. When, however, fleas have partaken of a full meal, they may suffer themselves to drop from an animal when this is resting in a spot suitable for them for their habitation. Thus fleas may be found in the nests, not only of rats, but of birds that may also harbour Pulicidæ. Probably they proceed from such spots to renew acquaintance with their hosts. In a preceding paragraph grounds have been adduced in support of the conclusion that, under certain climatic conditions, involving high temperature, they will leave the animal on which they occur, even although the latter does not exhibit sickness. In the case of the rat-flea (***Pulex cheopis***), there are grounds for concluding light possesses a similar influence.

VIII.—FLEAS ASSOCIATED WITH RATS AND MICE.

The Siphonaptera found associated with examples of the genus ***Mus*** are embraced in about thirty nominal species of fleas.

The following list thereof, together with an indication of the rat-species severally affected by these species, has been compiled from the works of Dr. O. Taschenberg, Carl F. Baker, and other writers on the group :—

Rat Flea Species and their Hosts.

—	*Mus rattus.*	*Mus rattus Alexandrinus.*	*Mus decumanus.*	*Mus musculus* (Mouse).	*Mus sylvaticus* (Mouse).	*Mus agrarius* (Mouse).	*Mus albipes.*	Miscellaneous.
Fam. Rhynchhoprionidæ.								
1. Rhynchoprion cæcata, *Enderlein*	×	...	...	...	...	...	...	
2. Argopsylla rhynchopsylla, *Tiraboschi* * ...	...	×	...	...	...	...	...	
Fam. Pulicidæ.								
3. Pulex irritans, *Lin.*	×	×	×	...	...	...	...	
4. Pulex pallidus, *Taschenberg*	...	...	...	...	...	...	×	
5. Pulex cheopis, *Rothschild*	×	×	×	...	...	...	...	*Mus gentilis*
6. Pulex phillipensis, *Herzog* †	×	...	...	...	...	...	...	
7. Pulex murinus, *Tiraboschi* ‡	...	×	×	...	...	...	...	
8. Pulex braziliensis, *Baker* §	×	...	×	...	...	...	...	
9. Ctenocephalus canis, *Curtis* ‖	×	×	×	...	...	...	...	
10. Ceratophyllus fasciatus, *Bosc.*	...	×	×	×	...	...	...	
11. Ceratophyllus londoniensis, *Rothschild* ...	× (?)	...	...	×	...	...	...	
12. Ceratophyllus gallinæ, *Schrank*	...	...	...	...	×	...	...	
13. Ceratophyllus pinnatus, *Wagner*	...	on Mus sp.			...	...	...	
14. Ceratophyllus italicus, *Tiraboschi* ...	...	×	×	×	×	...	...	
15. Typhloceras poppei, *Wagner*	...	...	...	...	×	...	...	
16. Ctenophthalmus agyrtes, *Heller*	...	...	...	...	×	...	...	
17. Ctenophthalmus assimilis, *Tasch.*... ...	...	...	...	...	×	...	...	
18. Ctenophthalmus proxima, *Wagner* ...	...	...	...	...	×	...	...	
19. Neopsylla bidentatiformis, *Wagner* ...	...	...	×	...	...	...	...	
20. Neopsylla pentacanthus, *Rothschild* ...	...	...	...	...	×	...	...	
Fam. Ctenopsyllidæ.								
21. Ctenopsyllus musculi, *Duges*	×	×	×	×	...	×	...	
22. Ctenopsyllus taschenbergi, *Wagner* ...	...	...	...	×	...	...	...	
23. Ctenopsyllus aganippes, *Rothschild* ...	...	on Mus sp.			...	...	...	
24. Stephanocircus dasyuri, *Skuse*	...	...	...	...	...	...	...	*Mus velutinus*
25. Stephanocircus simpsoni, *Rothschild* ...	...	...	...	...	...	...	...	*Mus velutinus*
26. Stephanocircus Thomasi, *Rothschild* ...	...	...	...	...	...	...	...	*Mus ferculinus*
Fam. Histrichopsyllidæ.								
27. Histricopsylla talpæ, *Curtis*	...	...	...	...	×	...	...	
28. Histricopsylla tripectinata, *Tiraboschi* ...	...	...	...	×	...	...	...	

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* This, when occurring on rats, has apparently been referred by some to the fowl flea, A. gallinacea.

† Pronounced by Rothschild to be identical with Pulex cheopis, *Rothschild.*

‡ Appears to be identical with Pulex cheopis. Report of Advisory Committee on Plague, 1st Report, "Journal of Hygiene" VI., 4, p. 425.

§ Appears to the writer to be also identical with the foregoing species.

‖ This is the flea that elsewhere in this report is styled Pulex serraticeps, *Taschb.* Probably the rat flea represents the form of this known as Ctenocephalus felis, *Rothschild*, a form that the writer has not the means of distinguishing.

In this treatment of the subject we are concerned only with fleas that have been met with on the species of *Mus* to which the first four columns of the foregoing tabulated statement relate, viz.:—*Mus rattus*, *Mus rattus Alexandrinus*, *Mus decumanus*, and *Mus musculus* (mouse). To these, then, have been allotted fourteen nominally different kinds, leaving out older synonyms that have not been mentioned.

There are grounds, however, for concluding that four of these are actually identical, viz.:—*Phillipensis*, Herzog; *murinus*, Tiraboschi; *braziliensis*, Baker; and *cheopis*, Rothschild (= *pallidus* auct. *née* Taschenberg); the last named, according to the rules of scientific nomenclature, being the earliest bestowed, superseding the others.

This, without reducing the number of species assigned to the division of *Pulex*, now known as *Ceratophyllus*, brings the total number of fleas recorded on the foregoing named rats and mice to eleven. Two of these, again, are Fowl Fleas, casually occurring on species of *Mus;* and further reference to them, therefore, may be omitted.

The census of flea-species met with in association with the rats directly related to man is thus as follows:—

1. Pulex irritans, *Lin.*
2. Pulex cheopis, *Rothschild.*
3. Ctenocephalus canis, *Curtis* (= C. felis, *Rotsch.*, and C. serraticeps).
4. Ceratophyllus fasciatus, *Bosc.*
5. Ceratophyllus londoniensis, *Rotsch.*
6. Ceratophyllus italicus, *Tiraboschi.*
7. Neopsylla bidentatiformis, *Wagner.*
8. Ctenopsyllus musculi, *Duges.*
9. Ctenopsyllus Taschenbergi, *Wagner.*

IX.—TABLE SHOWING DISTRIBUTION OF RAT-FLEA SPECIES.

[EXCLUDING AUSTRALIA.]

Species of Flea.	North and Central Europe, including Great Britain.	South Europe.	North Africa.	South Africa.	North Asia, including Japan.	South Asia, including British India.	Philippines.	North America.	South America.
I. Irritans, *Lin.* ...	...	Italy* occasionally (Tiraboschi)	...	...	...	Occasionally. *(Rep. Ind. Adv. Com.)	...	...	...
II. Cheopis, *Roths.*	Extremely rare, and then only on *Mus rattus* in Great Britain (Rothschild)	Marseilles, constituting 25 per cent. of fleas on ship rats (Gauthier and Raybaud) Genoa, constituting 40 per cent of fleas at the docks (Tiraboschi). Italy, generally — particularly frequent.	Egypt and Soudan (Rothschild)	Pretoria (Rothschild) South Africa, in boat from (Tidswell)	...	India—common Indian rat-flea: Constituting over 90 per cent. of the rat-fleas occurring at Bombay. (Rep. Ind. Adv. Com.)	Only flea found on rats. (Herzog) ...	Gulf Ports of U.S.A. and at Habana (Baker) ...	Brazil (Baker). Valparaiso, in large quantities (Rothschild). ...
III. Canis, *Curtis* ...	...	Italy — var. *murinus*, occasionally (Tiraboschi)	...	Pretoria (Rothschild)	...	India — occasionally. (Rep. Ind. Adv. Com.)	...	...	...
IV. Fasciatus, *Bosc.*	Usual flea on *Mus decumanus* in G. B., and apparently so in N. and Central Europe. On mouse in G. B.—uncommon. On the rare *Mus rattus* when met with (Rothschild)	South France, Italy, &c. (Tiraboschi)	...	Cape Town ... *Note.*—Only a few rat-fleas examined (Rothschild)	...	India — occasionally. (Rep. Ind. Adv. Com.)	...	...	...
V. Londoniensis, *Roths.*	Great Britain—London and Dover, on mice (Rothschild)	Italy (Rothschild) ...	...	...	...	...	...	...	Chili.
VI. Italicus, *Tiraboschi*	...	Italy (Tiraboschi) ...	...	...	...	...	...	...	...
VII. Bidentatiformis, *Wag.*	...	...	...	...	Siberia (Wagner)	...	...	...	...
VIII. Musculi, *Duges*	Great Britain—mice and rats, and general in N. and C. Europe	General	...	Pretoria ...	...	India	...	United States	Mexico.
IX. Taschenbergi, *Wag.*	Russia (Wagner) ...	...	...	...	...	...	...	...	...

X.—FLEAS ASSOCIATED WITH PARTICULAR SPECIES OF RATS IN AUSTRALIA.

[Queensland, Brisbane, &c.; New South Wales, Sydney; and West Australia, Perth.]

The following data are derived from a return made by Dr. F. Tidswell in 1904 (*cf.* Report on 4th Outbreak of Plague at Sydney, 1904, p. 4); from a communication from Dr. J. B. Cleland, Perth; and from H. Tryon's observations based on an examination of Rat Pulicidæ, submitted by the Commissioner of Public Health, Queensland (Dr. Burnett Ham), in 1902.

In the case of the West Australia return, it is assumed that all the fleas collected were derived from *Mus rattus*, var. *Alexandrinus*, for in a letter to Mr. C. Rothschild, a copy of which is to hand, Dr. Cleland refers to the Perth rats as being probably representatives of this rodent.

Table showing Relative Prevalency of Rat-flea Species on Different Kinds of Mus in Australia.

—	*Mus decumanus.*		*Mus rattus.*			*Mus musculus* (Mouse).
	Queensland (principally Brisbane), 1902.	New South Wales (Sydney), 1904.	Queensland (principally Brisbane), 1902.	New South Wales, 1904.	West Australia (Perth).	New South Wales (Sydney), 1904.
Pulex irritans, *Linn.*	·10%	...	...	...	...	...
Pulex cheopis, *Rothsch*	97%	88%	94%	38%	50%	34%
Ctenocephalus canis, *Curtis* [= P. irritans]	·2%	...	...	...	...	...
Ceratophyllus fasciatus, *Bosc.*	·5%	1%	...	23%	15%	17%
Ctenopsyllus musculi, *Duges**	2·2%	10·4%	6%	38%	48%	...
Number of fleas examined	943	86	49	13	66	35

* There are grounds for concluding that, owing to the defective method (as compared with that of Dr. Tidswell) pursued in securing the fleas occurring on Brisbane rats the percentage relating to this flea is much lower than it should be.

XI.—FLEAS OCCURRING UPON RATS IN AUSTRALIA.

The species of fleas that have been met with on the rats and mice associated with man in Australia are five in number, and are as follow:—

Pulex irritans, *Linn.*
Pulex cheopis, *Rothschild.*
Ctenocephalus canis, *Curtis* [=Pulex serraticeps].
Ceratophyllus fasciatus, *Bosc.*
Ctenopsyllus musculi, *Duges.*

Their relative frequency of occurrence, in connection with the rodents named, in the principal cities of the different States is shown in the following table, based, to some extent, on one formerly prepared by Dr. F. Tidswell, Microbiologist to the Board of Health of New South Wales (Report of the Board of Health on a Second Outbreak of Plague at Sydney, 1902, by J. Ashburton Thompson, M.D., &c., p. 72, 1903).

Distribution of Rat-flea Species in Australia and Relative Proportion of Occurrences.

—	Total Number of Fleas Examined.	*Irritans.*	*Cheopis.*	*Canis* (Serraticeps).	*Fasciatus.*	*Musculi.*
Queensland*...	1,609	·18%	90·8%	6% *a* [1·8%]	·37%	2·8%
Rockhampton	40	...	100%	...	...	...
Townsville	10	...	100%	...	...	...
New South Wales† (1902)	100	...	81%	1%	10%	8%
Sydney (1904)	134	...	69%	...	9%	23%
Newcastle	34	...	20·5%	..	3%	70%
West Australia‡ (Perth)	66	...	50%	*b*	1·5%	48·5%
West Australia, during epidemic period of year (December-July)	36	...	78%	...	...	22%
West Australia, non-epidemic period of year (July-December)	23	...	17%	...	...	82·6
Victoria§	80 or 90	Only one [it corresponds to Rothschild's description (of *P. cheopis*)] was non-pectinated (*i.e.*, all but one was *C. musculi* or *C. canis*).—[Dr. E. R.]				
Tasmania‖	290	·3%	1·4% *c*	A few	A few	Common Rat Flea.
New Zealand†	56	...	...	...	95%	5%

* Determinations by writers during 1902.
† Compiled from reports of Dr. Tidswell.
‡ Compiled from data furnished by Dr. J. B. Cleland.
§ Information supplied by Dr. E. Robertson.
‖ Compiled from data furnished by Dr. J. S. C. Elkington.

a The percentage 6 is arrived at by including a flea consignment of 378 examples, 93 of which were cat-fleas from Townsville. Rejecting this, the percentage is 1·8.

b Notwithstanding excessive prevalence of *Pulex canis* at Perth at some periods of year, Dr. Cleland made no reference to its occurrence on rats.

c "All *P. cheopis* were from two Hobart wharf rats (1 *rattus*, 1 *decumanus*) caught in the vicinity of the Sydney shipping berths in 1906."

From the foregoing tables it may be concluded that—

1. In Queensland (Brisbane, Rockhampton, and Townsville—also at Cairns), in New South Wales (Sydney, *not* Newcastle), and Western Australia (Perth), the rat-flea that preponderates is *Pulex cheopis.*
2. On the other hand in Victoria (Melbourne), Tasmania, and New Zealand, it is the least prevalent; the preponderating flea in the lastnamed colony being *Ceratophyllus fasciatus,* and in Victoria and Tasmania *Ctenopsyllus musculi.*

This varying numerical representation bears no relation to the kind of rat most prevalent, for in Tasmania and Western Australia—where, in both alike, the commoner rodent is *Mus rattus,* and *M. decumanus* is confined for the most part to the wharves—we have the rat-fleas that preponderate different ones.

The explanation of this is that *Pulex cheopis* is susceptible to meteorological influences to an extent that determines its distribution. This has been especially noted by the Hon. C. Rothschild, the great authority on *Siphonaptera,* who states that *Pulex cheopis* is "the common parasite of port and house rats in warmer portions of the earth. . . in the more temperate portions this *P. cheopis* is practically absent, being replaced by *C. fasciatus*" *in. litt.* (*cf.* Rothschild, C., "Journ. Hygiene", Vol. VI., 483-5, 1906). The data, set forth in the table entitled "Distribution of Rat-flea Species, Excluding Australia," indicates the effect of this factor in a very marked manner.

Again, there are grounds for concluding that climate may affect the ratio subsisting between the numbers in which *Pulex cheopis* are present on the same species of rats at different times of the year in the same place, in other words, its seasonal distribution. Thus on referring to the Table (p. 147) "Distribution of Rat-fleas in Australia, Relative Proportion of Occurrences," and Western Australia, therein, we find that during December-July the proportion between *P. cheopis* and *C. musculi* was 78 : 22, during July-December it was 17 : 86.

Dr. T. Burton Cleland in a letter to Rothschild adverting on this writes: "During October and half of November I got 4 of *P. cheopis* and 19 of *Tphlopsylla* (*musculi*). The odd ones collected for some months before were *P. cheopis,* 28, *Typhopsylla* (*musculi*), 8. This change in relative percentage is interesting if the smallness of the numbers does not (render it unreliable)."

In dealing with seasonal occurrence in treating of the Life History and Habits of Fleas (*vide* p. 140), similar variations in prevalence in different times of the year are mentioned.

It is unnecessary to pointout that the distribution of *Pulex cheopis,* both in time and place, has its strange counterpart in that of epidemic and, perhaps, also epizoötic plague.

XII.—THE RELATIONSHIP BETWEEN FLEA SPECIES AND RATS.

Observations indicate that not only do we find several different species of fleas comprised in the Flea Fauna of the rats especially referred to herein, but that we may encounter more than one kind of flea even on an individual rat.

In the course of this inquiry, the fleas secured on a single specimen of *Mus decumanus,* by the Commissioner, comprised examples of no less than four species—viz., *Pulex irritans,* 1 male; *Pulex cheopis,* 5 females; *C. canis* (*P. serraticeps*), 1 male; and *C. musculi,* 1 male.

The several flea species, when considered in relation to their hosts, exhibit, however, marked differences, in their degree of prevalence as well as in other features, as will appear from the subjoined facts.

Pulex irritans (The Human Flea).—The fact that of 1,609 fleas, derived from rats that came under examination, only 18 per cent. were representatives of this species will serve to indicate that its association with these animals is an exceedingly rare casual occurrence.

C. F. Baker in none of his memoirs on the *Siphonoptera* mentions *Mus* as serving as a host for this species; and this remark will, it is believed, be found to hold good also for other specialists on the flea group of insects. With the exception of references thereto in the Reports of the Indian Advisory Committee on Plague, that deal similarly with this exceptional incident, those who have written on the fleas of these animals in considering the subject of plague origination have very generally failed to remark this association.

Accordingly *Pulex irritans* cannot be regarded by any means as a normal rat-flea. It will not, indeed, thrive at all on the animals alluded to. The Indian Advisory Committee (Reports sect. XV., *vid.* "Journ. Hygiene," VII., 413) referring to an experiment designed to test this question state: "A great number of human fleas, caught in the manner we have already described (it may be objected that the method may account for the result.—H.T.), were put into a flea-proof cage along with a rat. Each day a census was made of the fleas still alive. After twenty-four hours it was found that only 1·2 per cent. of the fleas originally put in could be recovered, and after seventy-two hours this was further reduced to 1 per cent."

It may be added, again, that in the Queensland investigations no rat that had succumbed to plague and that was examined yielded a single specimen of this human flea species.

Ctenocephalus canis (=*P. serraticeps*) (The Dog and Cat Flea).—This flea is slightly more prevalent on rats than is the human flea proper. The fleas (1,609) examined yielded 1·8 per cent. that were representatives of this species (omitting reference to a parcel of rat-fleas from Townsville, in which—as there are grounds for concluding—fleas from different animal hosts had been incorporated). It was, moreover, mentioned in the report of the outbreak of plague at Sydney in 1900 ("Outbreak of Plague at Sydney, 1900." Report of the Chief Medical Officer, p. 40) that it was one of the fleas found upon the rats examined during that epidemic. It does not, however, appear to have been subsequently found on those examined in New South Wales in connection with later outbreaks.

The Indian Advisory Committee again refer to *Pulex serraticeps* as being occasionally found on the rats that came under their observations. C. F. Baker and other flea specialists do not mention any species of the genus *Mus* as a host for the dog and cat flea, and this may be regarded as a further indication that it seldom serves in this capacity, and cannot accordingly be considered as a normal rat-flea. Of course, the association of dogs and cats in identical spots with rats might alone suggest that the latter might temporarily harbour the fleas proper to these two animals.

Ctenopsyllus musculi (Rat and Mouse Flea).—This is exclusively a rat and mouse flea. It has never been met with on any other host—man included; nor has been recorded as living separate therefrom. It is probably the widest distributed of the rat-fleas, although, on account of its small size and habits as an ectoparasite, often overlooked. As has been already remarked, it formed 2·8 per cent. of the rat-fleas examined in Queensland. (Owing to the fact that the methods adopted here for securing rat-fleas are somewhat defective, this degree of prevalence is probably below what actually obtains.) Amongst New South Wales rat-fleas, as well as amongst those of elsewhere, it showed a greater prevalence. Being a blind insect, it might be expected that its attachment to its host would be more constant than would happen with fleas endowed with eyes, and this feature might explain also its inability to travel and so attach itself to other animals of different habit, when once occurring separate from a rat or mouse that had formerly harboured either it or its parent.

The fleas *Ceratophyllus fasciatus* and *Pulex cheopis* next to be considered may be especially regarded as rat-fleas, but insomuch as they are apparently susceptible to climatic conditions their geographical distribution is influenced by this circumstance.

With regard to Queensland, only ·37 per cent. of the fleas examined comprised *C. fasciatus*, and it thus occurred much less frequently than did the last-mentioned species (*C. musculi*). Moreover, it was not met with on any naturalised rat other than on *Mus decumanus*. On the other hand, in Sydney,* where the climate is, of course, more temperate, 10 of 134 fleas examined were this species, or 7·4 per cent. (Tidswell, "Report of the Board of Health on a Fourth Outbreak," p. 4, 1905.) In India, again, according to the report of the Advisory Committee, it is a flea only occasionally met with on rats. On the other hand, in north and central Europe, it is the common flea of rats (Rothschild), and is also a somewhat prevalent rat-flea in France (Marseilles) and Italy. A rat (*Mus Alexandrinus*) captured on a vessel that arrived in Brisbane in September, 1903, yielded eleven fleas that exclusively represented *C. fasciatus*. This was in striking contrast to the relative small proportion in which this flea figured at the time amongst rat-fleas yielded by Queensland Muridæ. In addition to its occurrence on rats and mice belonging to the genus *Mus*, this flea is recorded as occurring on the European mole (*Talpa europæa*), and on the additional Muridæ, *Cricetus cricetus* and *Eliomys quercinus*: whilst examples of a flea, obtained by Dr. T. L. Bancroft on the Australian Water-rat (*Hydromys leucogaster*), have been identified as representing a large variety of the same species. This varying host relationship indicates a faculty of accommodating itself to different conditions in this respect.

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* The rat-flea identified by W. W. Froggatt as *P. fasciatus* (*Vid.* "Report, Outbreak of Plague at Sydney, 1900," p. 40) and figured by this entomologist also as *P. fasciatus*, "Agr. Gaz.," N.S.W., May, 1901, Plate, fig. A, is *Pulex cheopis*.

Pulex cheopis (= *P. fasciatus*, Froggatt, and *P. pallidus*, Tidswell) (The Rat Flea of the Warmer Regions). The table setting forth the geographical distribution of rat-fleas will have indicated that this is *par excellence* the rat-flea proper of warmer climates taking the place of *C. fasciatus*, occurring where more temperate conditions prevail. We find it of extreme rarity in Great Britain (Rothschild), becoming very prevalent in ship rats at Marseilles, 25 per cent. (Gauthier and Raybaud); constituting 40 per cent. of the fleas on dock-frequenting rats in Genoa (Tiraboschi); and over 99 per cent. of those of town rats in Bombay. In the " New World " we note the same to happen as we proceed northwards from Argentina beyond the equator, large numbers have been received from Valparaiso, and what is evidently the flea under consideration is mentioned by C. F. Baker as being a common rat-flea in the Southern Gulf ports and in Havana.—(" Science," *n.s.*, Vol. XXII., p. 342, 1905.)

The tabulated statement, indicating the geographical distribution of rat-fleas in Australia, will again show us that *Pulex cheopis* is absent from New Zealand; practically non-occurring in Tasmania and Victoria (Melbourne); becoming prevalent in New South Wales (Sydney)—constituting there 62 per cent. and 87 per cent. respectively of rat-fleas in two instances when the matter was inquired into—and Western Australia (Perth); and very prevalent in Queensland, 90·8 per cent. of a total of 1,609 rat-fleas examined here being examples of this species. Moreover, with regard to the rats of this State of the Commonwealth, it may be added that every individual rat, which harboured fleas that were submitted for identification, harboured *Pulex cheopis*. In several instances, moreover, it exclusively occurred on the flea-infested rodent, especially when this was *Mus rattus*.

Again, special collections of rat-fleas, comprising few individuals, submitted for examination as coming from Rockhampton, Townsville, Cairns, and Brisbane, by the Bacteriological Institute, and by Drs. Tyrie and O'Brien respectively, were found to consist of *Pulex cheopis* alone.

XIII.—RAT-FLEAS ATTACKING BOTH RATS AND MAN.

Pulex irritans (The Human Flea).—This, as we have seen, is a flea of accidental occurrence on rats. It has been shown that it will bite rats, subsist on their blood, but that it will not thrive on this diet.

Ctenocephalus canis (*Pulex serraticeps*) (Dog and Cat Flea).—A casual rat-flea again. It commonly attacks man under stress of hunger (*vid.* " Habits of Fleas"), and may, it is considered, under these circumstances make its first meal on man. When very numerous, as sometimes happens, and its proper hosts are wanting, or wanting in sufficient numbers, it may prove in this respect very annoying. Its bite, nevertheless, is less severe than is that of *Pulex irritans*. It is, however, not partial to man. Thus when exceedingly prevalent on both dogs and cats, and increasing in numbers in their presence from the eggs that are shed on all sides from their flea-ridden bodies, and so living—for a while at least—at large, man may escape its attacks, or almost escape them. The occurrence of *Pulex serraticeps* on a rat, and its passage therefrom to man, must be an exceedingly rare contingency.

Ctenopsyllus musculi.—It is not in evidence that this rat and mouse flea has ever been met with on man, nor has it been demonstrated experimentally that it will even bite him. Tiraboschi (1903) even states that it shows positive distaste for the skin of man.

Ceratophyllus fasciatus.—The former portion of the preceding statement, regarding the habits of *C. musculi*, is believed to hold good with respect to this species also. However, both MM. Raybaud and Gauthier, working at Marseilles ("Comptes Rendues de la Société de Biologie," LIV., p. 1496, December, 1902—quoted by Dr. A. Thompson), and Dr. F. Tidswell, at Sydney (Dr. F. Tidswell "Ectoparasites of Rats. Report Outbreak Plague, Sydney, 1902," p. 72), have shown that this flea will, under experimental conditions, bite man, thus meeting an assertion by Nuttall and Galli Valerio to the contrary. Tiraboschi, writing in 1903, stated, however, that the Italian variety would not bite human beings.

Pulex cheopis (The Rat-flea).—We have in this species a flea that is more closely related structurally to the human flea than is any other of those mentioned, and that this affinity may extend also to its habits might be presumed. Moreover, we have an indication that it is especially able to adapt itself to feeding on different hosts, on considering the list of those on which it occurs. These, according to Rothschild, are *Alactaga (Dipus) jaculus* [Dipodidæ], *Acomys witherbyi, Arvicanthis-testicularis, Dipodillus watersi, Gerbillus robustus, Mus gentilis, M. decumanus, M. rattus, M. musculus* [Muridæ], and *Genetta dongolana* [Viverridæ]; and to these must be added the domestic rabbit.

Accordingly it is not surprising, therefore, that Cap. W. Glen Liston, Indian Medical Service ("Plague Rats and Fleas—Bombay Nat. His. Soc.," 24th November, 1904, "Indian Med. Gaz., 1904," and "Separatum, 1905"), should report that of thirty fleas caught on the human inhabitants of a house in Bombay no less than fourteen were rat-fleas (*Pulex cheopis*). It must be added, however, as a matter of great significance, that previous to this discovery—it was during the occurrence of a plague epidemic—the rats in a block of houses that contained this one were dying in large numbers, and that when the deaths ceased the inhabitants of these several houses became infested by rat-fleas.

. The Indian Advisory Committee (*vid.* Reports, XX., "A Note on Man as a Host of the Indian Rat-Flea," "Journ. Hyg.," VII., 472-476, July, 1907) has recorded that not only were examples of *Pulex cheopis* found to infest man under the conditions of its experiments, but that in houses in Bombay, during the epidemic of 1906, that were presumably plague-infected, abundant fleas of this species "were taken on the legs of men who entered the rooms only for a short time." Captures of 51, 34, and 60 individuals under these circumstances being mentioned in three instances referred to.

We must not, however, overlook the experience of equal significance that, when the special condition be absent—*i.e.*, when rats are not being or have not been decimated by plague—the partiality of this special rat-flea for man is not manifested. Thus Liston, whilst narrating what has been attributed to him, states that of 246 fleas caught on man in the absence of plague only one was a rat-flea.

The fleas occurring on man in Queensland have not been made an object of study, but it may be affirmed with confidence that under ordinary circumstances these comprise almost exclusively the Human Flea (*Pulex irritans*) and the Dog and Cat Flea (*Ctenocephalus canis*, or *Pulex serraticeps*). Dr. F. Tidswell, in his investigations regarding plague occurrence at Sydney, had occasion to inquire into this latter subject, and found that of ninety-seven fleas captured on man sixteen were *C. canis* and eighty-one *Pulex irritans* ("Report on Second Outbreak," p. 74). At the same time under the circumstances, referred to above as special, can it be doubted that the Bombay experience is not that also of Australia?—*i.e.*, that when an epizoötic amongst rats occurs or has just occurred rat-fleas leaving their proper host will find a host in man.

But not only do individuals of *Pulex cheopis* occur on man, but they will also feed upon him. MM. Raybaud and Gauthier (*l.c.*) and Dr. F. Tidswell ("Report, 1902," p. 75) experimenting independently in 1902 with, on the one hand, Sydney representatives of this species, and with Marseilles ones on the other, proved that they would bite human beings. The Indian Advisory Committee has further shown, to quote its report: "From the above facts (those detailed) it is seen that we were able to keep Rat Fleas (*Pulex cheopis*) alive for nearly four weeks by feeding them on human blood. There was a considerable mortality during this time, but the main facts stand out—(*a*) that they feed readily on man, and (*b*) that some of them (restricted to this diet) were still alive after twenty-five days, and (*c*) that rat-fleas, kept under similar circumstances but without food, never survive longer than a week." ("Report XX., Journ. Hyg.," VII., No. 3, July, 1907.)

The association of Rat Fleas (*Pulex cheopis*) with man has its exact analogue in what has been shown to occur over and over again with respect to guinea-pigs (*vid.* "Reports Indian Advisory Committee" *passim*)—normally they do not harbour rat-fleas; but when rat-fleas have access to them during the course of an epizoötic they may harbour them in large numbers, and commonly do so.

XIV.—FLEAS OF RATS IN RELATION TO THE BACILLUS OF PLAGUE.

"The blood of plague-infected rats may contain an enormous number of plague bacilli, even as many as 100,000,000 per cubic centimetre. An insect sucking the blood of most rats shortly before death would imbibe a large number of bacilli." ("Reports Indian Advisory Committee," sect. VIII., "Journ. Hyg.," VI., p. 23, 1906.)*

Plague Bacilli in Stomachs of Fleas.—The Japanese investigator Ogata found in 1897 that fleas from infected rats contained bacteria in their stomachs ("Centralbl. f. Bakteriol," Vol. XXI., p. 744).

In the following year, 1898, Dr. P. L. G. Simond found bacilli morphologically identical with those of plague in the stomachs of Indian Rat-fleas, that had fed upon plague-infected rats and mice. ("Ann. de l'Instit. Pasteur," XII., p. 625.)

Dr. F. Tidswell, in 1900, in the course of his Sydney investigations, on examining fleas from rats sick of plague found that they yielded bacilli that he microscopically determined to be plague bacilli. ("Report Outbreak Plague, Sydney, 1902," p. 56. Appendix A, Bacteriological Report.) At the same time he made several negative observations, results capable, however, of being explained since septicemia of plague is not always accompanied by marked numerical development of bacteria.

Dr. Buchanan, in 1902, found enormous numbers of plague bacilli in the stomach and other parts of the body of a flea (? *C. fasciatus*) taken from a rat dead of plague at Glasgow. ("7th Ann. Rep. Loc. Gov. Bd., Scotland, 1902," p. 73.)

Plague Bacilli Multiplying in Stomachs of Fleas. Also Occurring in their Fæces.—The above findings, repeated by these several observers, might have been regarded without much significance for Dr. Nuttall had observed, as the outcome of a long series of experiments, that when fleas partook of blood harbouring the bacteria of anthrax, chicken cholera, and some other diseases—not including those of plague, however—the bacteria lost their virulence after entering the flea stomach. (A. Thompson, "Report N.S.W., 1902," p. 75, referring to John Hopkins, "Hosp. Reports," VIII., p. 21, "Jl. Trop. Med.," V., p. 65.)

However, Zirolia found in 1902 that this did not apply to the bacillus of plague, for (to quote the "Report of the Advisory Committee," No. 1) "*Bacillus pestis* multiplied in the stomach of fleas and retained its original virulence for 7-8 days, during which time it was passed in the fæces" (*cf.* "Der Pest Bacillus in Organismus der flöhe," Centralbl. f. Bakteriol.," Vol. XXXI., p. 686.)

This observation was repeated by E. H. Hankin, who, writing in 1905, stated as confirmatory of the fact that the plague bacillus multiplies in the stomach of the fleas, "The other half of the stomach was subject to microscopical examination. The only microbes visible were bacilli with rounded ends, identical in appearance with those of plague. These were arranged in clusters of about a dozen individuals each, and appeared to be embedded in the tissues of the stomach wall. No bacilli were observed in the liquid contents of the stomach. The arrangement of the bacilli in clusters obviously suggests that they were engaged in reproduction *in situ*." (Hankin, E. H., "Epidemiology of Plague," "Journ. Hyg.," Vol. V., Jan., p. 81, 1905.)

The Indian Advisory Committee after conducting a series of experiments to demonstrate the relation between plague bacilli and the Rat Flea (*Pulex cheopis*) conclude:—(1) That the flea imbibing blood of plague-smitten animals may imbibe plague bacilli; (2) that the average capacity of the flea stomach is 0.5 c.m.; (3) that a rat-flea imbibing blood from a plague-infected rat might receive as many as 5,000 germs into its stomach; and (4) that fleas feeding on a large proportion of plague-infected rats just before death would imbibe some bacilli. ("Report, sect. XV., Journ. Hyg.," VII., 3, p. 397, July, 1907.)

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* For an account of the mechanism of the mouth organs of the Rat Flea (*Pulex cheopis*) and of its mode of working, *vid.* "The Physiological Anatomy of the Mouth Parts and Alimentary Canal of the Indian Rat Flea." ("Reports Indian Advisory Committee," III., "Journ. Hyg.," vi., 486-495, Pl. IX., Sep., 1906.) "The blood is sucked up from the wound made by the pricking apparatus of the insect through the mouth into the pharynx; it is passed down the pharynx and œsophagus by a successive wave of contraction of the muscles from before backwards: from the œsophagus it passes into the stomach, at the anterior end of which is a valvular arrangement called the gizard." *ib.* "Soon after a flea has filled its stomach absorption begins, and the blood in the stomach is greatly reduced in volume, finally becoming a thick tarry mass" (*ib.* sect. VII.) "The blood, after the end of the digestive process, in the stomach passes into the rectum as a thick, slimy, dark red mass, and appears at the anus as minute dark red tarry droplets." *ib.* The flea has the habit, moreover, "of squirting bright red blood from the anus during the operation of sucking," constantly diluting, therefore, the contents of its alimentary tract.

The same investigators have also found evidence of multiplication (*i.e.*, development) of bacteria in the rat-fleas' stomach in the following circumstance: When the rat-flea is transferred from a plague-infected rat to a healthy one it imbibes additional blood for some days onwards—but bacteria free blood; and, notwithstanding the contents of its alimentary tract are presumably diluted and even washed out in this way, "for the first four days after fleas have had an opportunity of feeding on septicæmic blood 43 per cent. of them contain abundant plague bacilli in their stomachs, and that from the 5th to the 12th day 13 per cent. are still infected." (Report, p. 402.)

The same was found to occur in the case of the human flea when fed on plague bacilli infected blood. "Fleas with large numbers of plague bacilli were found up to the 4th day"—and "the proportion of infected fleas gradually diminished from the 2nd to the 4th day."

No involution forms of plague bacteria were met with by the Advisory Committee, or by previous observers, in the stomachs of fleas that had fed on plague-infected rats, the organisms being the ordinary ones staining bipolarly. This is in harmony with the fact of the bacilli undergoing active development—(Report, "Journ. Hyg.," VII., p. 398)—and of their living under suitable nutritive conditions.

[NOTE.—Observations made by the Government Bacteriologist, C. J. Pound, *vid.* Appendix ("Experiments to test the possible transmission of plague from one animal to another by the agency of fleas"), on the contrary have lead him to recognise involution forms as a feature.]

Further, as a significant fact, the Advisory Committee found that apparently a peculiar influence of season on the numerical development of plague bacilli in the stomach of rat-fleas took place. Thus those secured from this source during a season when epidemic plague obtained were sixteen times more numerous than during a non-epidemic season. Moreover, in the non-epidemic season no flea was found with plague bacilli in its stomach after the 7th day since it fed on plague-bacilli-free blood.—("Report, sec. XV., Journ. Hyg.," VII., p. 404.)

The question as to whether the flea in the larval and pupal stages of its life may harbour the plague bacillus is still a matter for investigators.

The fleas mentioned as ones whose stomachs have contained plague bacilli, when the fleas themselves have fed on septicæmic rats, are the Rat Flea (*P. cheopis*) and the Human Flea (*P. irritans*). It evidently is the case also with *C. fasciatus* and *C. canis* (*P. serraticeps*). Apparently no evidence suggestive of the multiplication of the plague bacillus is forthcoming in the case of the latter two.

XV.—RAT-FLEA THEORY OF DISSEMINATION OF BUBONIC PLAGUE.

Introductory.

It may be regarded as a generally accepted fact that there is a connection between plague epizoötics (rat plague) and plague epidemics (human plague). "A relationship in time and place between plague in rat and plague in man."

A great deal of evidence tends to show that with the exception of what takes place in pneumonic plague, and plague contracted through the alimentary tract, this connection is not generally due to contagion or infection, &c., but results from inoculation, which the plague rat, acting alone, is, of course, unable to bring about.

Hankin, in 1898, on considering the epidemiological data relating to plague occurrence at Bombay in 1898 first came to the conclusion that—(1)"The incidence of plague on neighbourhoods and in houses stood in relation to their accessibility to rats," and (2) "that some intermediary insect was necessary to communicate the infection from rat to man." ("Annales de l'Institut Pasteur," 1898, quoted by Dr. A. Thompson "Report Plague, Sydney, 1902," p. 58.)

Dr. P. L. G. Simond, a French investigator working in India, thereupon formulated the "flea-hypothesis" to account for the inter-communication of plague between rat and man, and for the dissemination of plague by this agency. (*Vid.* Simond, P.L., La Propagation de la Peste, "Ann. de l'Inst. Pasteur," Vol. XII., p. 265, 1898.)

Simond united the Bed Bug (*Cimex lectuarius*) with the flea in this hypothesis; but Nutall, in his experiments, in which the latter insect figured, obtained only negative results ("John Hopkins' Hospital Reports," Vol. VIII., pp. 17-21).*

In Queensland, the connection between plague in rats and plague in man has from the outset been regarded as indicating the main lines of procedure in checking the course of the epidemic, and already in 1902 the study of the flea fauna of rats was inquired into as possibly suggestive of a subsidiary procedure.

In New South Wales, the Chief Medical Officer of the Government, Dr. Ashburton Thompson, when confronted with a plague epidemic in Sydney, in 1900, at once espoused the Rat-flea Theory, and in the reports of that and the succeeding years adduced a good deal of circumstantial evidence tending to show possible inoculation by the flea (Reports, 1900, pp. 23, 40, 56-7, pars. 262-271, 272-292, 293-294, 298-299; but nothing of a direct character). On epidemiological grounds alone, he regarded the existence of such an intermediary as indispensable. However, the Bacteriological Investigator, Dr. Tidswell—one of the first to recognise a special rat-flea, that he provisionally but erroneously referred to *Pulex pallidus* of Taschenberg—considered that it was the Dog and Cat Flea (*Pulex serraticeps*) that there were grounds for implicating as the agent in the inter-communication of the disease from rats to man (*vid.* "Report, Plague, Sydney, 1902," p. 71).

Plague Transference from Rat to Rat or to other Animals by Fleas.

Of course, if it can be shown that rat-fleas, or any one kind of rat-flea, commonly communicates plague from the rat to any other lowly animal (*e.g.*, guinea-pig) it lends colour to the suggestion that plague may also be communicated by a similar agency from rat to human beings, and *vice versâ*.

Dr. P. L. G. Simond, experimenting in 1898—(1) produced plague by inoculation of animals with bacilli from fleas that had fed on plague rats, and (2) found that if he placed a plague sick rat in a large jar, and put a healthy rat or mouse in the same jar, so as to exclude possibility of contact, but not other means of infection, and left or placed fleas on the sick individual, the previously healthy rat or mouse sometimes died of plague when the sick one on its demise was allowed to remain in the jar with it for some hours. His experiment has been regarded merely as consistent with the flea hypothesis only. Simond used in his investigations the cat-flea. ("Annales de l'Institut Pasteur," October, 1898.)

Dr. Tidswell, experimenting in 1900, produced fatal plague in guinea-pigs; he employed an emulsion from Rat Fleas (*Pulex cheopis*), the only ones in which he had found the plague bacillus.

Dr. J. S. C. Elkington, in the course of his investigations (*vid.* "Austr. Med. Gaz.," XXII., p. 348) in the Plague Research Laboratory, Bombay, in 1902, repeated Simond's experiments, obviating, however, the conditions that rendered them inconclusive. In this case "all communication between animal and animal, both direct and indirect, was prevented except that which was the subject of investigation"—*i.e.*, communicability by fleas. The results secured were positive. The flea he experimented with was the special Rat Flea (*Pulex cheopis*). It has been remarked by Ashburton Thompson that whilst he (Dr. Elkington) thus confirmed Simond's hypothesis he did not, of course, establish the correctness of his inference." ("Report Outbreak, Sydney, 1902," p. 75-6.)

From the foregoing statements it will appear that in each of the positive results mentioned the rat-flea has been Pulex cheopis.

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* Herzog, in 1904, found *Bacillus pestis* in three human lice (*Pediculus capitis*) taken from a child which had died of plague in Manila. This patient had cervical buboes, and Herzog suggests that injection might have been conveyed by the lice.—["Report Advis. Com.," Pt. I.; "Journal Hygiene," VI., pt. 4, p. 430, 1906.] It is needless to indicate that such a method of conveyance cannot generally take place. Yersin, moreover, already in 1895 had met with virulent plague bacillus in flies. (Chez les mouches mortes.) [La peste bubonique *Annales de l' Institut Pasteur*, IX., 1895, p. 589.] These insects, however, can play but little part in plague of the bubonic type arising from inoculation.

With regard to other rat-frequenting fleas, the following may be added:—

Dog and Cat Flea (*C. canis*, or *Pulex serraticeps*).—Tidswell, experimenting in 1902 (*vid.* "Report Outbreak, Sydney, 1902," pp. 73-4) with examples of this flea species derived from a dog—these fleas having first fed on a fatally plague-smitten rat—ascertained that, although they actually bit the guinea-pigs entering into the investigation, they failed to communicate plague to it. The experiment was repeated with identical results.

The Indian Advisory Committee, during the present year 1907, experimented again with every precaution adequate to secure positive results, but without obtaining them. "Twenty-seven experiments to transmit plague from animal to animal by means of Cat Fleas (*Pulex felis* = *Pulex serraticeps*) were made; none of these were successful," however. The animals used were again the highly plague susceptible guinea-pigs, and in each case about twenty fleas from septicæmic rats were placed upon them.—("Reports, sect. XV., Journ. Hyg.," VII., p. 412-3, 1907.)

The Rat Flea of Temperate Climes (*Ceratophylus fasciatus*).—On the other hand, two experiments that they conducted showed definitely that this flea could convey plague from infected rats to healthy guinea-pigs ("Op. Cit.," p. 414).

The Human Flea (*Pulex irritans*).—Again the Advisory Committee have found that this flea of man, when fed on septicæmic rats, communicates plague to healthy guinea-pigs; but sparingly. In thirty-eight experiments "three of the guinea-pigs became plague-infected." This is explained also by the fact that human fleas will neither thrive on guinea-pigs nor on rats ("Op. Cit.," p. 413).

We are thus impelled to the conclusion, derived from experimental data, that plague may be communicated from animal to animal by two rat-fleas especially (*Pulex fasciatus* and *P. cheopis*). The data are too limited to indicate that in the case of the former this dire disease is commonly conveyed by it. As, however, it is not a flea that occurs on rats, except quite rarely, within regions subject to endemic plague, we may for the present dismiss it as not being a factor in this connection of much significance.

XVI.—PLAGUE INFECTION BY FLEAS—THE PROCESS.

The method of infection by fleas may now be alluded to.

The Indian Advisory Committee have enumerated the possible ways in which this transmission may take place (Report XV., "Journal of Hygiene," VII., 3, p. 415), and by experimental inquiry ascertained which of these is most likely to be realised under natural conditions of plague occurrence.

(1.) *Ingestion of Infected Fleas.*—In the several hundred instances in which animals under test conditions had become infected by fleas, no case of primary mesenteric bubo was remarked, whereas when disease was brought about through the medium of infected food this feature was observed in no less than 70 per cent. of the plague occurrences, whence it is concluded that infection by the agency of fleas is brought about in some other way than the above.

(2.) *The Flea Proboscis when Accidentally Contaminated by Plague Bacilli.*—Plague infection by this agency has been suggested by C. F. Baker ("Science," *n.s.*, XXII., p. 342, 1905). Examining the proboscides of 100 infected fleas the Advisory Committee failed to demonstrate the presence of plague bacilli in any instance, but remark that "it is impossible to exclude this means in the case of animals which have recently left a septicæmic host, but it is significant that the largest number of infections occurred with fleas which had fed on infected animals within thirty-six hours" ("Op. Cit.," p. 416). Dr. Tidswell has shown that plague may be communicated by a plague-germ contaminated instrument, reproducing approximately the features of flea-bite.—("Report, Outbreak Plague, Sydney, 1900," App., p. 56.)

(3.) *The Flea-saliva Charged with Bacteria when Instilled into the Subject at the Time of Biting.*—No instance of the occurrence of the plague bacillus in the salivary glands of fleas, or, indeed, outside the alimentary tract, has been yielded by the dissection and examination of hundreds of fleas at the hands of the abovementioned investigators.—(Report, Advisory Committee, *l.c.*)

(4.) *Regurgitation of Stomach Contents of Flea at Time of Biting, and Infection of Flea-saliva, &c., thereby.*—The Indian Committee referred to have found no evidence to support this conclusion or even in favour of the view that regurgitation takes place, a process that the anatomy of the flea is not compatible with.—(Report, *l.c.*)

(5.) *Retention of Infected Blood in the Flea-pharynx, or Mouth Cavity, and its Subsequent Passage to the Subject of Flea-bite.*—The foregoing remark applies here also.

(6.) "*By the bacilli contained in the flea-fæces being deposited on the skin and then being either injected by the pricker or rubbed into the wounds made by the pricker.*"—("Op. Cit," p. 418.)

It was early shown in the case of the New South Wales Plague Investigations that when the surface of the skin of an animal was deprived of hair, so that in the shaving process the skin was abraded but not cut, it might be plague-infected at this spot by an artificial culture of the bacillus being smeared over it.—(Dr. Tidswell.)

Again, the Indian Advisory Committee has demonstrated conclusively "that the bite of a healthy flea affords a sufficient avenue for infection by septicæmic blood if it is spread upon the bitten part."—(Report, XV., "Journal Hygiene," VII., p. 419-420, 1907.)

In dealing with the Habits of Fleas, it has been already pointed out that when feeding they both deposit fæcal matter, as well as droplets of bright blood, on the sites of their biting operations (*vid.* p. 139).

Surgeon-General Wyman (Marine Hospital Service, U.S.A.) in his "Report on Bubonic Plague, 1900," writing on the subject of plague communication, stated that, as compared with other methods for securing this:—"It is much more probable that the fleas or other insects having their habitation on animals deposit their dejecta, and in this way infect their bites. It is to be remembered that a very small abrasion may furnish a point of entrance for the virus, and this, too, may be made by the scratching on the irritation caused by insect bites" ("Op. Cit.," p. 25-6). The inference and the facts whereon it is based were, however, already mentioned by Dr. P. L. Simond in 1898.

Zirolia, as has already been seen, recorded in 1902 that the bacillus of plague passed from the body of fleas into their fæces, and the Advisory Committee, after confirming this discovery, have demonstrated, further, the possibility of infection by flea-bite when this bacillus-laden matter is already present at the site of injury; but it is careful in pointing out that it has been unable to ascertain whether this is the usual process.—(Report Advisory Committee XV.)

Dr. P. L. G. Simond, in the course of his Indian observations in 1898, detected plague bacilli in some of the phlyctenules (blebs arising from flea-bites) occurring on Indian Plague subjects, and Dr. Ashburton Thompson also noted the same in a phlyctenule, that he regarded as arising from flea-bite, on a person suffering from plague at the very outset of his investigations in 1900 of plague occurrence in New South Wales.

The Indian Advisory Committee has further stated that when guinea-pigs have naturally succumbed to plague under conditions that suggest rat-flea infection, and rat-fleas are moreover found upon them, phlyctenules may occur.—(Report, XIV., "Journal Hygiene," VII., 385.)

XVII.—RAT-FLEA (PULEX CHEOPIS) ESPECIALLY IMPLICATED IN CONVEYING PLAGUE FROM ANIMAL TO ANIMAL, AND INFERENTIALLY FROM RAT TO MAN.

The circumstantial evidence on which the "Flea Theory" was based was further strengthened by Glen Liston, in 1905. This investigator showed in 1904, as the outcome of investigations conducted in Bombay, that—(1) There was one flea infesting rats in India far more commonly than did any other—viz., *Pulex cheopis*, Rothsc.; (2) that these fleas when feeding on a plague-rat harboured the plague bacillus in their bodies, and that it multiplied therein; (3) that it passed from one animal to another—*e.g.*, rat to guinea-pig—on the bacillus-infected flea being transferred or transferring itself; (4) that where fatal plague occurred many of these bacillus-infected fleas were at large, that they thereupon might gain access to guinea-pigs (whereas the latter only become flea-infested during an epidemic) with possible fatal results (plague); and (5) that after a local epizoötic (rat plague) man was also found to harbour these Rat Fleas (*Pulex cheopis*) and might become plague-infected as had the guinea-pigs. (*Vid.* Liston, W. G., "Plague Rats and Fleas," "Journ. Bombay Nat. Hist. Soc.," XVI, p. 253-274, 1905.)*

Special Investigations in India.—The conveyance of plague by a special rat-flea—viz., *Pulex cheopis*—from rats to other animals has, however, been proved in a very definite manner by the Advisory Committee on Plague, that has during the past two years been carrying on its investigations in India, and in Bombay especially. There the rôle enacted by this flea became obviously a matter for inquiry, since it constituted 99 per cent. of the rat-flea fauna.

These "Reports on Plague Investigations in India" are published in the "Journal for Hygiene" of September, 1906 (Vol. VI., No. 4) and in subsequent issues. The following will serve as a summary of the experiments alluded to:—

In the first instance, healthy rats were confined in close proximity to ones that, inoculated with plague, had succumbed to that disease, and that previous to this had been artificially infected with Rat Fleas (*P. cheopis*), the separate confinement of the two rats in each case being so arranged that both contact with and access to all excreta were excluded, although it was provided that these fleas could pass from the inoculated to the healthy rat; whereupon it was found that a passage of fleas to the healthy rat took place, that in many cases these fleas contained plague bacilli that were capable of originating the disease, and that when the healthy rat thus infested was not likely to be immune it died of plague to the extent of 69 per cent., this extent of infection then falling to 38 per cent. when partly immune rats of local origin were employed.

That the plague had originated in the healthy rats through the intermediary of these rat-fleas was further demonstrated by the fact that when they were actually transferred from artificially plague-infected to healthy rats, it resulted to the extent of 61 per cent. of instances when healthy English rats were used, and to from 47 per cent. to 66 per cent. when Bombay partly immune *Mus decumanus* rats were employed.

Further, on constructing a series of miniature houses (godowns), so as to reproduce the conditions pertaining to ordinary domiciles, it was found that whenever these were so constructed so as to admit of rats to their roofs, but not to their interiors, guinea-pigs confined therein became successively infested by rat-fleas and infected by plague, but that in those houses to which rats could not gain access plague was originated in guinea-pigs living therein, either by transferring rat-fleas to them, derived from plague-infected guinea-pigs, or on accidental admission of rat-fleas from other sources. Also, that when so confined, guinea-pigs had under these conditions died of plague; healthy flea-free guinea-pigs, subsequently introduced, became plague smitten, and that the contagion remained in the place in proportion as the test animals were accessible to, and were found to be infested with, fleas. In other words, that "if the fleas be present, the epizoötic, once started, spreads from animal to animal, the rate of progress being in direct proportion to the number of fleas present. Further, that when in one of the houses, to the interior of whose roof fleas could not gain access, healthy guinea-pigs were confined, guinea-pigs became flea-infested and infected when running on the ground, to a less extent when the cage was placed 2 inches therefrom, and not at all when it was suspended 2 feet above it. The fact of infection taking place when rats were located 2 inches above the ground indicates that contact with infected soil is not necessary for plague to originate, and that "an epizoötic of plague might start without direct contact of healthy with infected animals."

To demonstrate that this communication of plague from guinea-pigs to guinea-pigs was through the intermediary of fleas, rat-fleas were taken from a morbid guinea-pig and allowed to feed through muslin on healthy animals, with the result that the fact of the establishment of the disease in the manner suggested was indicated by the positive outcome of the experiment.

The state of things that occurred in actual domiciles in which plague existed or had existed was next inquired into, advantage being taken of the fact that plague-susceptible guinea-pigs would serve as hosts for and for the collection of fleas.

Guinea-pigs free from fleas were introduced into rooms in which persons had died of plague, or from which plague-infected rats had been taken and allowed to be at large in these for periods of from eighteen to twenty-four hours. And these guinea-pigs not only collected the fleas on their bodies, most of which were rat-fleas, but 29 per cent. of them contracted plague, and died of plague within a few days after being restored to ordinary confinement. As before, the fleas that they yielded in many cases harboured plague bacilli in their stomachs, and were capable of infecting additional animals.

Further, after first washing the floors and walls of the rooms with an acid solution of perchloride of mercury, and so adequately disinfecting them for plague, but not for fleas, and then introducing guinea-pigs, these latter became plague-infected if rat-fleas were present.

That the infection was actually due to fleas was shown by the positive results of collecting fleas from rats occurring in plague-infected houses, and transferring them to healthy rats or guinea-pigs in the laboratory, when these in due course became infected by plague and died.

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* This extension of the Rat-flea Theory is usually associated with the name of Liston and referred to as "Capt. Liston's Rat-flea Theory" (*vid.* "The Indian Med. Gaz.," XLI., No. 7, 295-298, July, 1906). It was announced in a paper read at a meeting of the Bombay Natural History Society held in November, 1904. However, on 22nd July, 1903, Dr. J. S. C. Elkington, in an Address on "Fleas and Plague Convection" read before the Victorian Branch of the British Medical Association, has advanced an almost identical theory, adducing at the same time interesting experimental data. It appears, however, from his statement that his work was done at least partly in association with Liston. "While in India I was able to make, partly in association with Captain Liston, I.M.S., at the Imperial Plague Research Laboratory, Bombay, some observations upon their habits that seemed to further confirm these insects in the rôle of plague distributors." ("*Op. Cit.* Separate," p. 5.) Apparently subsequent writers have regarded Dr. Elkington's announcements in the light of "news in advance" and have accordingly passed them over. Dr. Tidswell, however, had much earlier (1900) directed attention to a special flea (*Pulex pallidus*, now known to be identical with *P. cheopis*) as occurring upon rats during the prevalence of plague in Sydney, and was alive to the possible significance of this circumstance. However, he appears to have been deterred from sustaining his views later by experimental data. (1) Since at the time he was working rat-fleas were no longer obtainable, and (2) since *Pulex cheopis* was to be dismissed as being a flea "not occurring on man habitually."—(Report, Sydney, 1902, p. 71.)

Similarly fleas taken from the clean guinea-pigs allowed to run in plague-infected houses, and transferred to fresh animals, communicated plague to them in 8 out of 40 experiments.

In the next place, plague-free white rats, guinea-pigs, and monkeys were placed in enclosures, which precluded contact as well as soil-infection, in plague-infected rooms, pairs of one animal or another being used in each of the forty-two experiments of this class conducted, one individual being confined to a flea-proof receptacle, and the other to an adjacent one accessible to these insects (one animal being thus a control). In the latter case plague resulted in four instances, or 10 per cent. gave positive results.

As a variation of the same experiments, the enclosures for individual animals, whilst being such as to protect them from soil or contact infection, were surrounded, as a screen to fleas, by 2½ inches of "tanglefoot," or were unprovided with this protection, the "tanglefoot" being replaced by sand. (Twenty-nine experiments were conducted.) In the latter case the animal became infested with fleas, one having as many as twenty; seven became fatally infected by plague. In the former individual fleas only were found on three of the rats, and no animals became plague-infected.

Examining the fleas entrapped, 247 in number, it was found that 147 were human-fleas, 84 were rat-fleas, and 16 cat-fleas. Moreover, a large proportion of each kind were examined. No plague bacilli were found in the cat-fleas, 1 only in 85 of the human-fleas, and no less than 23 out of 77 of the rat-fleas harboured plague organisms.

It was also shown that, when rats in the course of an epizoötic died of plague, the pathological features manifested in their bodies corresponded to those exhibited by artificially rat-flea infected animals, and hence it was inferred that in Nature and under experimental conditions the animals had alike succumbed to a single agency. This identity especially related to the site in which buboes arose that in both instances, where the place of inoculation could be observed, was nigh thereto.

XVIII.—THE FLEA HYPOTHESIS AND THE PLAGUE EPIDEMIC.

The correctness of the inference underlying the "Flea Hypothesis," that epidemic plague arises from the infection of man by rats already subjected to the disease through the intermediary of rat-fleas, and—to a preponderating extent—through that of a particular species, *Pulex cheopis*, can, of course, be alone proved by an heroic experiment still to be conducted.

The hypothesis, however, receives the strongest confirmation from the fact already set forth, that plague, not only can be, but is, actually conveyed by this agency from one animal to another, and when the plague-rat is its original source especially. Particularly is this the case when it is remembered, as demonstrated in the course of the investigations of the Indian Advisory Committee already referred to, that the recipient animal is one belonging to the great group of vertebrates that includes man—that of the Primates—and the victim is "our immediate ancestor," the monkey.

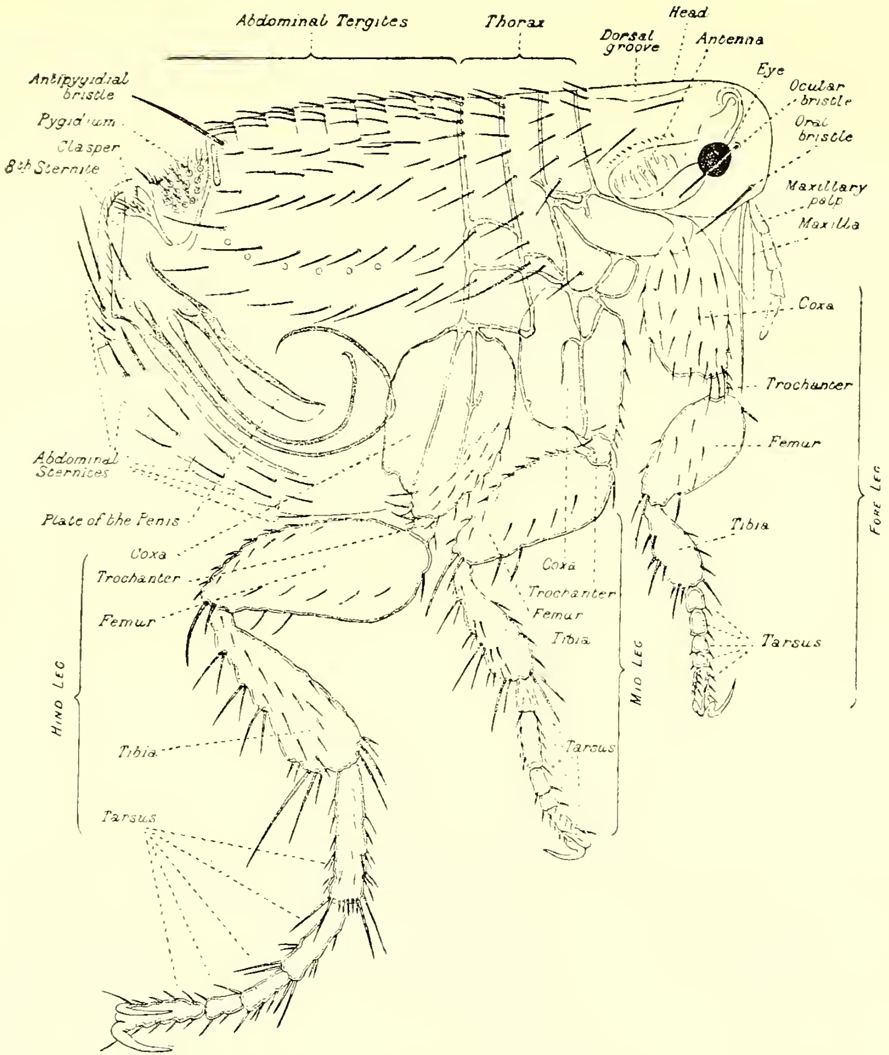
But, notwithstanding this lack of direct evidence—attesting the correctness of the hypothesis—as applied to man only we find, as in other quarters, confirmatory evidence of a very pronounced kind when considering plague from an epidemic standpoint.

This phase of the subject has been more conveniently treated of in the previous Section of the Report, and it will suffice, therefore, to direct attention to the light that the Flea Hypothesis sheds on the otherwise anomalous features connected with—(1) *The range of occurrence of epidemic plague (geographical distribution)*; (2) *the range of its occurrence in time (seasonal distribution)*; (3) *latent plague infection and inherence of plague infection in clothes, &c., notwithstanding the low vitality characterising the plague bacillus itself*; (4) *recrudescence of plague where plague incidence due to fresh importation is to be excluded as a cause*; (5) *absence of place infection properly so called apart from the presence of the "plague-rat"*; (6) *the occurrence of epizoötic (rat-plague) and epidemic plague (human plague) successively*; (7) *certain local conditions of plague occurrence, and other incidents coming within the purview of the epidemiologist.*

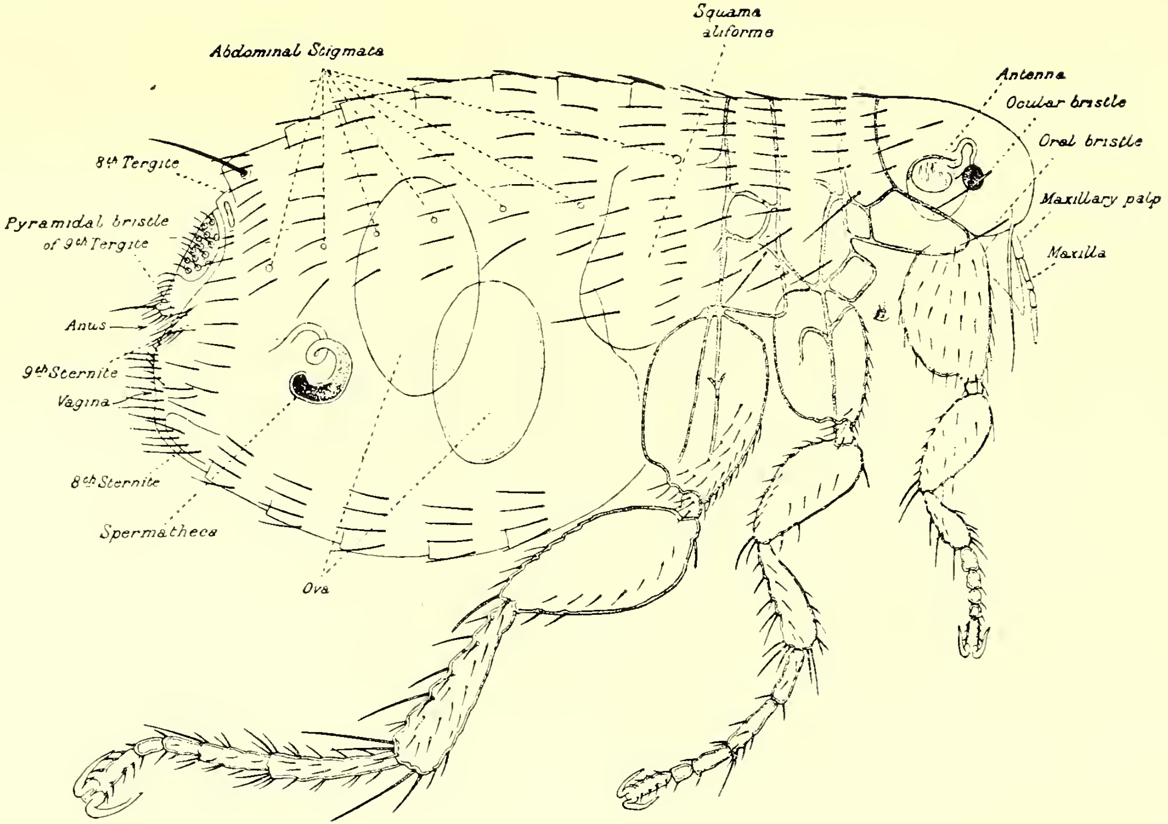
XIX.—RAT-FLEAS.

Their Distinguishing Features.

In treating of rat-fleas, it may serve a useful purpose to indicate briefly the distinguishing features of those met with more commonly on our naturalised species of *Mus*. The entire flea fauna of the members of this genus is probably not yet known, and accordingly the reader must consult the systematic works on the *Siphonaptera*, by Rothschild, Taschenberg, Baker, Wagner, and others to ascertain the nomenclature and affinities of all the flea-species on members of this group that he may encounter.



♂ PULEX CHEOPIS, *ROTHSCHILD.*



♀ PULEX CHEOPIS, *ROTHSCHILD.*

D. A. Turkhud, M.B., Edin.

E. Wilson, Cambridge.

THE RAT-FLEA.

From Report on Plague Investigations in India issued by the Advisory Committee. ("The Journal of Hygiene," Vol. 7, No. 3, July, 1907.")

The common kinds alluded to are the Human Flea (*Pulex irritans*); the Dog and Cat Flea (*Ctenocephalus canis*); the Rat Flea of plague regions (*Pulex cheopis*); and the Rat Flea prevalent elsewhere (*Ceratophyllus fasciatus*).

As these again have been dealt with in detail from the comparative standpoint in readily accessible *Reports on Plague, already published, the treatment now bestowed on the subject need be none other than a brief one.

When the identity of fleas only is in question, examination may be readily effected, if, after being superficially dried (not to the extent of admitting shrinkage with the consequent inclusion of air spaces), the specimen be soaked in Xylol, and then examined under a low power of the microscope whilst in this medium and beneath a cover-glass.

Attention in the course of this examination should be given to the following features:—Relative size, colour, eyes, comb-like teeth on head and prothorax, bristles on hind-border of head, in orbital circle, on granulated plate (pygidium), and on posterior extremity above.

The species of rat-fleas above referred to may then be incidentally recognised by reference to the following:—

Tabulated Arrangement of the Commoner Rat-fleas.

Fleas without eyes	*Ctenopsyllus musculi* (and its allies)
Fleas with eyes	A
A. Fleas without comb (*Ctenidium*) on head or prothorax	B
Fleas with comb (*Ctenidium*) on head or prothorax	C
B. Brown, or dark-brown, with few bristles only on hind border of head (usually only one present, that on the posterior inferior angle); ocular bristle inferior; antipygidial bristle short; claws large	*Pulex irritans*
Red-brown, with continuous row of bristles on hind border of head; ocular bristle superior; antipygidial bristle long; claws smaller	*Pulex cheopis*
C. Comb present on both head and prothorax... ..	*Ctenocephalus canis* (*P. serraticeps*)
Comb present on prothorax only	*Ceratophyllus fasciatus.*

By reason of the interest that attaches to the rat-flea of epizoötic and epidemic plague (*Pulex cheopis*, Rothschild; syn. *Pulex pallidus*, Tidswell, *nec* Taschenberg), the following technical description by Rothschild is subjoined:—

The Plague Rat Flea (*Pulex cheopis*, Roths.). *Vide* Plate III.—"This species is larger than *P. nubicus*, the palpus being shorter than the rostrum, and not reaching to the end of the coxæ. In the male, sternites three to seven inclusive, bear four bristles, whilst those of the female have five. The hind femur bears, in addition to the lateral series of hairs, two subventral bristles before the apex. The first segment of the mid-tarsus is rather less than two-thirds the length of the second, while that of the hind tarsus is about three-quarters as long again as the second segment. The long apical bristles of the 2nd segment of the hind tarsus reach to the middle of the 5th segment in the male, and not quite so far in the female. The 4th segment is as in *P. nubicus*. The 8th sternite bears two long bristles before the end on each side, and numerous short ones besides.

"The anterior process of the clasper of the male (cl.) is compressed, being symmetrical in shape. The upper or anterior edge is convex, bearing along this edge a number of rather long bristles. The second process (cl.) of the clasper is slender, with a few short hairs at its end. The 9th sternite (St.) gradually widens towards the apex. The plate of the penis (p.) is curved upwards and pointed at the end." (*See* Plate III.)

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* (1) Report on Plague, Sydney, 1902; (2) Dr. J. S. C. Elkington, Address on Fleas and Plague Convection, July, 1903; (3) "Journal of Tropical Medicine," IX., June, 1906; and (4) Report, Advisory Committee, "Journal Hygiene," VII., July, 1907.

Male and Female Fleas Contrasted.

As the special features of fleas are emphasised in the male individuals, and as a knowledge of the distinction between the sexes is otherwise useful to the investigator, we may contrast them.

The sexes of fleas are distinguished especially by features presented by the hind-body. (Examining this from above and regarding the scute that occupies an emargination in the hind-borders of the thorax (*metanotum*), but that does not form a complete ring, as the 1st segment, and counting backwards to the hindermost, or 8th segment, we find—should the insect before us be a *male*—that the dorsum, or tergite, of this closely resembles those of the preceding six segments regarded individually, that they are all somewhat abbreviated and pressed together, and that thus the united anal and sexual openings find an outlet above. Also, that jointed to the variously modified 9th abdominal segment (on which occurs the pygidium), that constitutes a firm support for them, are two prominent wing-like bristle-bearing plates that exhibit different outlines in different flea-species, and are named claspers. Two less conspicuous and retractile claspers may, too, be observed below these. Again, should the specimen be rendered transparent, as when mounted as a miscroscopical object, the interior of the hind-body will exhibit three chitinous and more or less internal convoluted flattened rods, that have for their purpose the support of the peculiar male-organ proper.

In the *female* flea, on the other hand, the dorsum (tergite) of the 8th hind-body segment is very much enlarged and projects downwards on each side so as to encroach on the ventral scute (sternite), and so render it almost lanceolate in outline. The tergites of the six preceding segments again are not abbreviated and pressed together. Accordingly in this sex the hind-body as a whole is longer and more or less terminally swollen and jointed instead of terminating above in an angle. Moreover, there are no paired organs or claspers attached to the 9th segment, although there exists a terminal abdominal style. Within the hind-body may be discerned a chitinous seminal vesicle—the spermatheca. And, finally, in comparison with the male insect, the female is of larger size.

XX.—RAT PARASITES OTHER THAN FLEAS.

(1) THE BED BUG (*Cimex lectuarius*).—During the outbreak of plague in Brisbane in 1900, and to some extent in subsequent ones, examples of this insect were occasionally encountered on rats. This was remarked also by the New South Wales investigators with regard to Sydney rats during the 1902 epidemic. There the bugs occurred in all stages of development on their temporary hosts, and were apparently as numerous thereon subsequent to the course of the epizoötic as whilst this was in progress. As has been already stated, Dr. Simond, in 1898, associated this insect with the flea when propounding his theory to explain the mode of communication of the disease from rats to man in epidemic plague. The possibility of infection being brought about in this way was discredited by the direct experiments of Dr. Nutall subsequently conducted (*vid.* John Hopkins, Hospital Reports, VIII., p. 17-21), and the negative results of these have been now generally accepted.

(2) LOUSE (*Hæmatopinus piliferus*, Burmeister).—Two examples of a louse closely resembling this dog-parasite were comprised in a small collection of fleas from an example of *Mus decumanus*. Zirolia has been previously cited as an authority for the statement regarding the apparent conveyance of plague from man to man in one instance by the Human Louse (*Pediculus capitis*). Nothing in the present connection is, however, to be inferred from his observations.

(3) MITE (*Holaspis sp.* [Fam. Gamasidæ]).—Twelve examples of this acarus were found amongst the ecto-parasites of a specimen of *Mus decumanus*, procured by the Commissioner in Brisbane. It was also the exclusive external inhabitant of an indigenous rat obtained in the Herbert River district. Moreover, an apparently related mite has been encountered by Dr. T. L. Bancroft on a Brisbane specimen of the native Water Rat (*Hydromys leucogaster*). Its occurrence on rats may be accounted for by the fact that it is predatory on insects, and may be even partial to rat-fleas.

B. BURNETT HAM,
Commissioner of Public Health.

APPENDIX A.

BACTERIOLOGICAL REPORT.

By C. J. Pound, Government Bacteriologist.

I.—PLAGUE DISSEMINATED AMONG RATS THROUGH THE AGENCY OF FLEAS.

The following episode is interesting in connection with the transmissibility of plague among rats by means of fleas, and occurred during the early part of the third epidemic of plague in Brisbane.

Mr. A. brought a half-grown live rat in a cage to the Institute. The rat was removed from the wire cage and placed in another larger one with nine perfectly healthy and nearly full-grown rats. On Mr. A. being handed back his cage he incidentally mentioned that a member of his family had only a few days previously been removed as a plague patient to the Plague Hospital; he further stated that it might be of interest to know if anything was wrong with the rat that he brought, as it had been caught in his house.

Although the rat in question appeared quite lively and well, I instructed the caretaker to inform me at once should any sign of sickness appear, either in this or in any of the other rats in the same cage. Four days later, two of the rats were found dead, and three others were sick, including the rat that Mr. A. brought. These three died the next morning. *Post-mortem* and bacteriological examination revealed the fact that all five rats had died of plague; moreover, on the bodies of two of the rats fleas were found, and in the bodies of several fleas plague bacilli were discovered.

In view of the results of this investigation it was decided to destroy the remaining five rats, several of which were in a dying condition. The box was drenched inside and out with strong carbolic acid solution, and then placed in a fumigating room, and subjected for about six hours to ammonia and then to sulphurous acid gas.

On examination, each of these rats was found to be infected with plague, and in four of the ten rats examined there was a marked hæmorrhagic condition of the subcutaneous tissues. The nine healthy rats referred to had been reared at the Institute and belonged to one family, being born about twelve months previous. They were always in a healthy condition, and on no occasion whatever were fleas or other parasites found upon them. It was, therefore, quite evident that the fleas which had been introduced with Mr. A.'s rat were solely responsible for the outbreak of plague among the nine healthy rats, although it was quite possible that Mr. A.'s rat may not have been infected at the time it was placed with the others.

It should be mentioned that the plague patient, a young boy, the son of Mr. A., had been for several weeks in the habit of capturing rats (handling them without any precaution) for the purpose of obtaining the capitation fee of 6d. per head offered by the Department.

II.—INTERESTING OBSERVATIONS IN CONNECTION WITH THE POSSIBLE ACCIDENTAL TRANSMISSION OF PLAGUE FROM RAT TO RAT BY THE AGENCY OF COCKROACHES.

The facts in relation to this case are as follow:—In a room specially set apart for keeping all inoculated animals are two large stands with wide shelves, on which are placed long lead-lined trays about 2 inches deep, containing carbolic solution. Standing in these trays and surrounded by the carbolic solution are the various strong glass jars in which are kept the experimental animals. These jars are about 16 inches high and about 9 inches in diameter. Each jar contains only one animal—guinea-pig, rat, or mouse, as the case may be—and is covered with a mosquito-proof fine wire gauze lid. On the occasion referred to, in November, 1902, a healthy guinea-pig that was being kept as a control for certain experiments suddenly became sick, and after three days it died. *Post-mortem* and bacteriological examination proved that this guinea-pig had died from a generalised form of plague, but no lesion were found to indicate that it had been infected through the skin. A careful examination revealed the fact that in the zinc binding of the wire cover there were several very young cockroaches. These were promptly destroyed. On examination of the covers of the other adjoining jars, some of which contained plague-infected guinea-pigs and rats, more young cockroaches were discovered.

It is quite evident that these young cockroaches had become hidden in the zinc lining of the covers when the jars were not in use, and standing on the shelf alone, unprotected by the tray of carbolic solution.

As soon as a jar was occupied by an experimental animal the young cockroaches that had been hidden from view in the zinc lining during the day time would, after dark, crawl down the inside of the jar and feed on the animals' food. Apparently before their presence was discovered, some of these young cockroaches had fallen from a jar containing a plague-infected animal into the carbolic solution, and then swam either to the jar containing the healthy guinea-pig or to the side of the tray, and then hid away in the cover of an empty jar. In any case, it was more than probable that the food had become contaminated with bacilli.

In order to ascertain whether the cockroaches had anything to do with the transmission of plague, a healthy guinea-pig was placed in a sterilised jar covered with the usual wire lid, but whose zinc lining was free from cockroaches. The jar was placed on the shelf, but not in a tray. In the course of a few days young cockroaches made their appearance, and as usual lived during the daytime in the zinc lining. Eventually the guinea-pig sickened and died of plague.

There was sufficient convincing evidence in this one observation to show the extreme danger which existed when insects like cockroaches once gained access to places where animals are kept, after being infected with such a virulent disease as plague.

After this, the whole of the building and everything, such as shelves, benches, jars, &c., were subjected to thorough and repeated disinfection, and all holes and crevices carefully closed. The result is that no cockroaches have been seen since, and, although every day during the past eight months numbers of plague and healthy guinea-pigs and rats have been kept in the same jars and standing in the same trays, no symptoms of the disease has appeared in any animal unless specially infected.

III.—DISCOVERY OF TRYPANOSMA IN BRISBANE RATS.*

On several occasions during the microscopical examinations of specimens of blood from suspected plague rats, numerous motile micro-parasites were met with, and sometimes they were associated with plague bacilli, although there is apparently no connection between the two. These organisms belong to the protozoa, and are known as Trypanosoma; moreover, they are identical in every respect with those described by Lewis and Crookshank, and found by them as existing in fully 25 per cent. of the London sewer rats *(Mus decumanus)*. So far, I have only found them in the same species of rats in Brisbane. Compared with bacteria they are very large, varying in size from the one-six-hundredth to the one-four-hundredth part of an inch in length, and about one-three-thousandth part of an inch in breadth. They stain readily with fuchsine, gentian-violet, methylene-blue, and Bismarck-brown, but not by Gram's method. When viewed under a twelfth homm. imm. objective, the stained organism is seen to have a tapering body. One end has a spine-like process, and the other terminates in a long whip-like flagellum, while along nearly the whole of the body is an extremely delicate undulating fur-like membrane. The body has a granular appearance, with one, two, or three bright refractive spherules. In the living condition the organism is usually seen pushing its way between the blood corpuscles. Like all organisms possessing only one flagellum, it invariably progresses flagellum first. At times the organism becomes apparently attached to a red corpuscle, and lashes its flagellum in all directions, in such a manner as though endeavouring to set itself free. Occasionally it will turn round and round on its long axis, or it will progress rapidly backwards and forwards for a short distance only.

In some specimens of blood, besides the free swimming organisms, others may be seen in various stages of development encysted in the red blood corpuscles; therefore, like the malarial fever parasites, they spend part of their life history inside the cellular elements of the blood.

The point of interest about this organism is, that it is very closely allied to the trypanosoma only very recently discovered by Dr. J. E. Dutton of the Liverpool School of Tropical Medicine. A few weeks later Drs. Manson and Daniels also found the same micro-parasite in the blood of patients who were suffering from irregularly intermittent attacks of fever, accompanied by loss of condition, considerable lassitude, and debility.

Similar organisms have been found in the blood of cattle and horses suffering from an extremely fatal disease known as Surra and Nagana in India.

IV.—EXPERIMENTS ON THE INGESTION OF INFECTIVE MATERIAL *(B. PESTIS)* BY GUINEA-PIGS, RATS, AND MICE.

Experiment 1.—Six guinea-pigs were fed with bran mixed with *B. Pestis* from spleen of plague rat, and one (a control) inoculated with the same material.

Result: Two died of plague within six days, one became sick but recovered, and the other three remained unaffected. The inoculated control died in four days.

Experiment 2.—Six rats, all caught at one time in an apparently healthy locality, were used for three separate experiments as follows:—One rat fed with lung of plague guinea-pig, one control rat inoculated with some of the same material.

Result: The fed rat remained alive, and the control died of plague in six days.

Experiment 3.—One rat was fed with spleen of guinea-pig that had died of plague. One rat inoculated with same material.

Result: The fed rat died in seven and a-half days. The inoculated one in five days.

Experiment 4.—One rat fed with kidney of guinea-pig that had died of septicemic plague. One rat inoculated with the same material.

Result: The fed rat became very sick, but recovered. The inoculated one died of plague in four and a-half days.

Experiment 5.—Five rats fed with young Agar culture of plague bacilli, and one rat inoculated with the same material.

Result: The fed rats remained unaffected. The inoculated one died of plague in five days.

Experiment 6.—Two mice were fed with *B. pestis* from spleen of guinea-pig that had died of plague, and a mouse inoculated with the same material.

Result: Those fed remained unaffected, while the inoculated one died of plague in three days.

Experiment 7.—One mouse fed with *B. pestis* from serum culture, and one inoculated with same material.

Result: Both died of plague within four days.

These experiments show that rats are not nearly so susceptible to plague by ingestion as by inoculation of the infected material. It is, therefore, possible that under natural conditions the disease is not so readily contracted by healthy rats feeding on the bodies of plague-infected rats as is generally supposed.

Plague Bacilli in Fæces and Urine.

During the last outbreak of plague a quantity of rat fæces was collected from a warehouse in the city where a number of plague-infected rats had been found. The fæces in question were thoroughly broken up, mixed with sterilised water, and strained through fine linen. Some of the mixture was then injected into three guinea-pigs, with the result that one died of plague in five days, the second became sick and recovered, and the third remained unaffected.

Considering the difficulty to find evidence of gastro-intestinal infection among the rats submitted for examination, this experiment has only an indirect bearing on the possibility of plague bacilli being present in the intestinal tract of some naturally infected rats.

On one occasion only *B. pestis* was detected by means of culture and animal experiment in the urine of a rat which had recently died of plague.

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* A like discovery was made in 1888 by Dr. T. L. Bancroft [*cf.* Bancroft (T. L.) "Note on Hæmatomonas in Rat's Blood."—*Proc. Roy. Soc. Qd.*, V., p. 31-2, Brisb., 1888].

V.—EXPERIMENTS TO DEMONSTRATE THE PRESENCE OF PLAGUE BACILLI IN THE URINE AND FÆCES OF ANIMALS SUFFERING FROM PLAGUE.

URINE.

1. Guinea-pig inoculated with urine collected from human bubonic plague case. Result: Remained alive.

2. Guinea-pig, control to above, inoculated with lymph from gland of the above plague case. Result: Died in 5 days. Plague bacilli numerous in all organs.

3. Guinea-pig injected with urine collected from plague rat. Result: Died of plague in 4½ days.

4. Rat injected with urine from plague guinea-pig. Result: Died of plague in 5 days.

FÆCES.

1. Two rats were injected with an emulsion of fæces in sterile water from a septicæmic plague rat. Result: Both died of plague, one in 4 days and the other in 5 days.

2. Guinea-pig injected with fæces from a plague-infected rat. Result: Died of plague in 4½ days.

In connection with these observations it is somewhat remarkable that, in all the experiments on guinea-pigs, &c., with rats' excreta, which was found in a dried condition in many produce stores where plague rats were continually being discovered, it was shown that in no single instance did any of the animals, rats, or guinea-pigs that were inoculated with these fæces die or become infected with plague.

VI.—THE EXAMINATION FOR PLAGUE BACILLI OF PRODUCE, ETC., FROM PLAGUE-INFECTED PREMISES WHERE BOTH HUMAN SUBJECTS AND RATS WERE FOUND INFECTED WITH PLAGUE.

A piece of recently imported Chinese matting was thoroughly washed in sterilised water, which was afterwards centrifugalised and the sediment injected in equal proportions into two rabbits, two guinea-pigs, and two rats. Result: One guinea-pig died from accidental causes within 24 hours, the other animals all remained alive, without showing any signs of illness.

Two guinea-pigs injected with centrifugalised washings of chaff obtained from floor of a plague-infected produce store. Result: Remained alive; no signs of sickness.

Guinea-pig (533) inoculated with earth from around the entrance to a rat burrow from which plague rats had been captured. Result: Died in 8 days from tetanus.

Guinea-pig (549) fed with chaff and corn floor sweepings from infected produce store. Result: Remained alive, with no signs of sickness.

Three mice (607-9) placed in jar with quantity of chaff floor sweepings from plague-infected produce store. Result: All remained alive, and showed no symptoms of illness for nearly three months.

Two guinea-pigs (610-611) placed in a jar with a quantity of the same chaff. Result: Remained alive, and showed no signs of sickness for over two months.

Guinea-pig placed in a jar with supposed infected chaff. Result: Remained alive; no signs of sickness.

Guinea-pig inoculated with infusion of supposed infected chaff sweepings. Result: Died in 1½ days of sepsis. No *B. pestis* detected.

Two guinea-pigs injected with washing from bananas from infected stores where a number of plague rats had been found. Result: One died of sepsis in 2½ days. No *B. pestis* detected. The others remained alive without showing any signs of sickness.

Four guinea-pigs, each placed in a separate glass jar with a quantity of chaff (floor sweepings) from plague-infected premises. Result: Remained perfectly well, without showing any signs of illness.

Four guinea-pigs inoculated with infusion of chaff (floor sweepings). Result: One died of sepsis in 36 hours; the other remained alive.

These experiments demonstrate the difficulty in recovering plague bacilli from material where there is every reason to believe plague-infected rats have been running and contaminating the same with urine and fæces.

VII.—EXPERIMENTAL FEEDING OF VARIOUS LABORATORY ANIMALS WITH VIRULENT PLAGUE BACILLI.

Throughout these experiments the material used was, unless otherwise stated, portions of a spleen of either a rat or a guinea-pig just dead of acute plague.

Before being fed to the various animals, the spleen was mashed up with a little pollard to the consistency of dough.

The animals experimented upon were rats, mice, guinea-pigs, rabbits, monkeys, fowls, and ducks.

All material, whether from spleen, liver, lung, or culture, was not, unless it contained an abundance of active bacilli, fed to the animals.

1. A rat (597), inoculated with virulent plague material from a naturally infected rat, was, in consequence of showing no symptoms of sickness, fed 17 days later with half the spleen of a plague rat. Result: Remained unaffected. Forty-four days from last special feeding it was refed with spleen of a plague rat and died in 4 days with general infection.

2. Four young rats, all belonging to one family, and about three-parts grown, were fed with bread well mixed with spleen pulp from a septicæmic plague rat. Each animal received an equal share. Result: One died in 2½ days, and another in 3½ days. The *post-mortem* examination showed general infection and *B. pestis* numerous in all organisms. The other two remained alive, eating well, and showing no signs of sickness. Seven days later both were inoculated with virulent plague bacilli from a naturally infected rat, and, again, they showed no signs of sickness. Twenty-seven days later both were fed with liver containing virulent bacilli from a naturally infected rat, but without producing any noticeable ill-effects. Thirty-nine days later they were both inoculated with plague material from a naturally infected rat. One died in 3 days, and the other in 6½ days. Both had general infection with *B. pestis* in all organs and glands.

3. Two rats (573-4) were fed with liver containing numerous *B. pestis* from a naturally infected rat. Result: One died of plague in 3½ days. The other, which remained in the same jar, did not show any apparent signs of sickness until the 12th day, when its coat became ruffled. Its temperature rose on the 13th day, it became gradually weaker, refused food, and died of plague on the 20th day.

4. Five young rats (698-702), all of one family, were each fed with an active virulent serum culture of *B. pestis* from a guinea-pig. Result: 698 died of plague in 5 days. The other four remained alive after eating their plague-stricken companion. Ten days later they were specially fed with spleen containing large numbers of virulent plague bacilli from a guinea-pig. Six hundred and ninety-nine died of plague in 3 days, 700 died of plague in 9 days, and 701 died of plague in 15 days, while 702 showed no apparent signs of sickness. Fifty-five days from first special feeding rat 702 was inoculated in the groin with *B. pestis* from a plague guinea-pig. It died of plague in 4 days. *B. pestis* were numerous in all organs and glands.

5. One rat fed with spleen of plague guinea-pig. Result: Died of plague in 5½ days.

6. A rat which had been inoculated with spleen blood of plague and had shown no signs of illness up to the 12th day was, 16 days later, fed with half a plague rat's spleen. Result: Remained alive, showing no signs of sickness.

7. Rat fed with virulent culture from plague guinea-pig. Result: Died within 24 hours. Accidental death. All organs apparently normal. No *B. pestis* detected.

8. Rat fed with serum culture direct from plague guinea-pig. Result: Remained alive, showing no symptoms of illness.

9. Two rats fed with spleen from plague guinea-pig. Result: One died of plague in 3 days, the other remained alive, without showing any signs of illness.

10. Rat fed with spleen of plague guinea-pig. Result: Remained alive.

11. Two rats fed with spleen of plague guinea-pig. Result: Both died of plague, one in 3 days and the other in 8 days.

12. Rat fed with spleen of plague rat. Result: Died of plague in 4 days.

13. Rat fed with spleen of plague guinea-pig. Result: Died of plague in 3 days.

14. Two rats fed with liver of plague rat. Result: One died of plague in 7 days, the other remained unaffected.

15. Two rats fed with liver of plague guinea-pig. Result: One died of plague in 5 days, and was partly eaten during the night by the other rat, which remained unaffected.

16. Rat fed with spleen of plague guinea-pig. Result: Died of plague in 6 days.

17. Native Water Rat (*Hydromys leucogaster*) fed with lung of plague guinea-pig. Result: Died of plague in 3 days.

18. Three mice fed with spleen of plague guinea-pig. Result: All remained unaffected.

19. Four mice fed with liver of plague rat. Result: All remained unaffected.

20. Guinea-pig fed with plague gland from human case. Result: Remained alive, showing no signs of sickness. Control guinea-pig inoculated with same material died of plague.

21. Guinea-pig fed with culture of virulent plague bacilli. Result: No signs of sickness.

22. Three guinea-pigs fed with spleen of plague guinea-pig. Result: No symptoms of sickness whatever.

23. Three guinea-pigs fed with liver of plague guinea-pig. Result: All remained alive. No symptoms of sickness.

24. Two rabbits fed with spleen of plague guinea-pig. Result: Both remained unaffected.

25. Two rabbits fed with spleen of plague guinea-pig. Result: Both remained unaffected.

26. Monkey fed with spleen of plague guinea-pig. Result: Remained alive. No signs of sickness.

27. Two chickens fed with spleen of plague guinea-pig. Result: Remained alive. No signs of illness.

28. Fowl fed with spleen of plague rat. Result: Remained unaffected.

29. Fowl fed with spleen of plague guinea-pig. Result: Remained unaffected.

30. Two ducks fed with spleen of plague guinea-pig. Result: No symptoms of illness whatever.

In another series of experiments, the following animals were each fed with a pure culture of plague bacilli:—Four rats, seven mice, five guinea-pigs, one monkey, and two ducks; and, for control purposes, one rat and one guinea-pig were inoculated. Result: All the fed animals remained alive, without showing any signs of sickness, while the control rat and guinea-pig both died of plague within 5 days.

Table showing summary of results of feeding various species of animal with virulent plague bacilli:—

Animal.	No. Fed.	Died of Plague.
Rats	33	19
Mice	14	...
Guinea-pigs	13	1
Rabbits	4	...
Monkeys	2	...
Fowls	4	...
Ducks	4	...
Total	74	20

Of the 19 rats fed which died of plague the following species were represented:—Brown rats (*Mus decumanus*), black rats (*Mus rattus*), reddish-brown rats (*Mus Alexandrinus rufus*), and native water-rat (*Hydromys leucogaster*).

There are several points of interest in these experiments. In the first place, it shows that rats, although the most susceptible of all animals to plague, are not so readily affected by ingestion of plague material as by subcutaneous inoculation. For, of 33 rats specially fed, 15 remained unaffected, and 18, or 54½ per cent., died of plague; whereas, in another experiment, where 35 rats were specially inoculated, only 6 were unaffected, while 29, or 83 per cent., died of plague.

Only one out of thirteen guinea-pigs was affected with plague.

Of the 14 mice, 4 rabbits, 2 monkeys, 4 fowls, and 4 ducks specially fed, not one was affected with plague, which demonstrates the comparative immunity to the disease when the *B. pestis* is ingested.

VIII.—EXPERIMENTS TO TEST THE POSSIBLE TRANSMISSION OF PLAGUE FROM ONE ANIMAL TO ANOTHER BY THE AGENCY OF FLEAS.

In a previous report I drew attention to an interesting observation, whereby 9 healthy rats, free from fleas, all became infected with plague through the introduction, by accident, of a plague rat that had fleas upon it into the same cage. It was, of course, open to argument that plague may have been transmitted to the healthy rats by some agency other than fleas, as, for instance, ingestion or inhalation.

In order to obtain further information on this subject, the following experiments were carried out:—

(A.) Inoculation of healthy animals with an emulsion of fleas taken from plague-infected animals, or the presumably infected flea introduced direct into the subcutaneous tissue.

(B.) Injection of water in which fleas from plague-infected rats had been washed.

(C.) The placing of fleas taken from infected animals upon healthy animals.

With few exceptions, all the fleas used were of the species *Pulex serraticeps*, taken from the laboratory dog.

In the third series of experiments, the fleas were kept in small glass tubes for from 24 to 36 hours, to make them hungry, after which they were placed on the back of a guinea-pig's head, which animal had been inoculated 3 days previously. When each flea had been observed to adhere firmly to the skin of its host for 3 minutes or longer, it was removed by means of a pair of forceps, and then allowed to fasten on the skin of the experimental healthy animal.

Series A.

1. From a naturally plague-infected rat (*Mus rattus*) a flea was removed, and, after its stomach contents showed numerous cylindrical forms of plague bacilli, it was introduced into the subcutaneous tissue of the groin of a guinea-pig.

Result: The animal became sick and died of acute plague in 3 days. *Post mortem* revealed congestion of the lungs, liver, spleen, and the supra-renal bodies. *B. pestis* numerous in all organs and glands.

2. Four fleas from a plague rat (*Mus decumanus*) were placed one each under the skin in the inguinal region of 4 guinea-pigs.

Result: One became sick on the second day, and died of acute plague in 3½ days. *B. pestis* were numerous in all organs. The other 3 animals remained unaffected.

3. Guinea-pig inoculated with an emulsion, in sterile water, of a flea recently removed from a plague guinea-pig.

Result: Remained alive without showing any signs of sickness.

4. Guinea-pig injected with an emulsion of water of a flea removed from a plague-infected rat.

Result: Remained unaffected.

5. A flea, in which no *B. pestis* was detected, although removed from a plague guinea-pig, was injected into a healthy guinea-pig.

Result: Remained unaffected.

Series B.

1. Guinea-pig injected with sterile water in which 3 fleas obtained from a plague guinea-pig had been washed.

Result: Remained unaffected.

2. The above 3 fleas were mashed up with sterile water, and the whole injected into a guinea-pig.

Result: Died in 5½ days. *B. pestis* numerous in all organs.

3. Guinea-pig injected with sterilised water in which a flea from a plague-infected rat had been washed.

Result: Died in 4 days. *B. pestis* in all organs and glands. It is of special interest to note that plague bacilli were found in the stomach contents of each of these fleas.

4. Rat injected with water in which 7 fleas from a plague guinea-pig had been washed.

Result: Died of acute plague in 6 days.

5. Three fleas (*vide* No. 1, Series C) were mashed up with sterile water and injected into a healthy guinea-pig.

Result: Remained unaffected.

Note.—No plague bacilli were found in the stomachs of these fleas.

SERIES C.

1. One rat (*Mus decumanus*) had placed upon it 3 fleas taken from a naturally-infected plague rat.

Result: Remained unaffected.

2. Five fleas from a plague-infected guinea-pig were placed upon a healthy guinea-pig.

Result: Remained unaffected.

NOTE.—No *B. pestis* were detected in the stomach contents of either of these fleas.

3. A rat (*Mus decumanus*) had placed upon it 2 live fleas taken from a guinea-pig just dead of septicæmic plague.

Result: Remained unaffected. Some time after this rat was fed with a rat's spleen full of *B. pestis*, and, 10 days later, it was inoculated with virulent plague material, but remained alive without showing any symptoms of illness.

This rat, which was caught at the end of an epizoötic, had evidently acquired immunity under ordinary conditions. Fourteen days after the last inoculation it was killed, and, on examination, the characteristic pigmented glands of a recovered animal were readily apparent.

4. One of the fleas, which was placed on this rat, had in its stomach active *B. pestis*, causing the death of a young guinea-pig 5 days after they were injected.

5. Three fleas, which had been allowed to feed on a plague guinea-pig, were placed on a healthy rat (*Mus decumanus*).

Result: Died of plague in 12½ days. Lungs congested and mottled. Spleen enlarged. *B. pestis* numerous in all organs and glands. *B. pestis* found in both fleas.

6. Two fleas from a plague guinea-pig were placed on a rat (*Mus decumanus*).

Result: Remained alive, with no apparent signs of illness. No *B. pestis* found in either flea.

7. Four fleas from a plague guinea-pig were placed on a wild mouse.

Result: Remained unaffected.

8. Six fleas from a plague rat were placed on a mouse.

Result: The mouse died of plague in 5½ days.

SUMMARY OF RESULTS OF EXPERIMENTS WITH FLEAS.

Of 9 rats and guinea-pigs inoculated with fleas or portions of fleas from plague-infected animals, 3 died of the disease, while the fleas that were placed on the other 8 animals were subsequently proved to have no plague bacilli either on or in their bodies.

Of 4 animals injected with sterile water, in which fleas from plague animals had been washed, only 2 died of plague.

Of 7 animals that had fleas placed upon them which had previously been allowed to live for about 2 days on a plague-infected animal, only 2 died of the disease, and 1 rat had acquired immunity under natural conditions to plague.

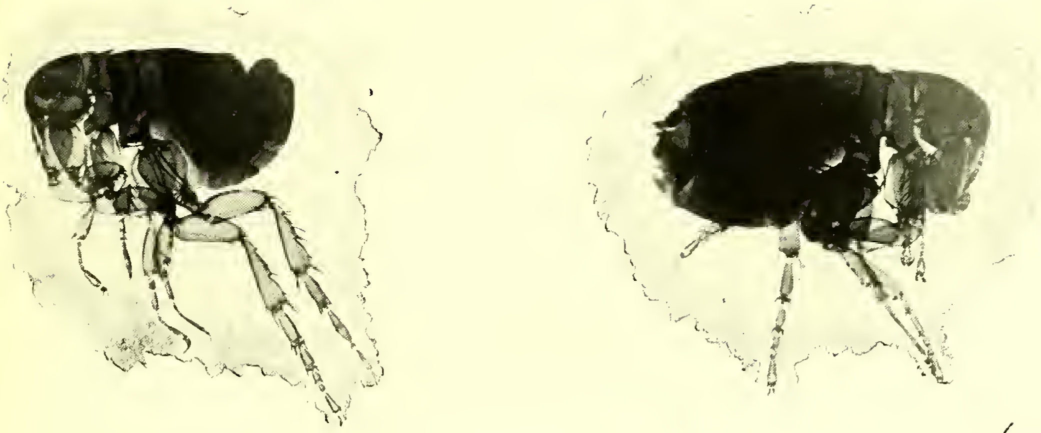
In the cases of the other 4, it was subsequently found that the fleas placed upon these animals were free from plague bacilli.

Price 9s.]

By Authority: GEORGE ARTHUR VAUGHAN, Government Printer, William street, Brisbane.

Plate IV.

PHOTOMICROGRAPHS OF FLEAS FROM BRISBANE RATS.

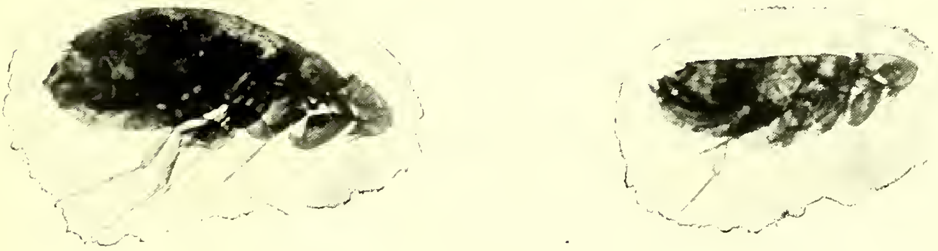


male × 18

female × 18

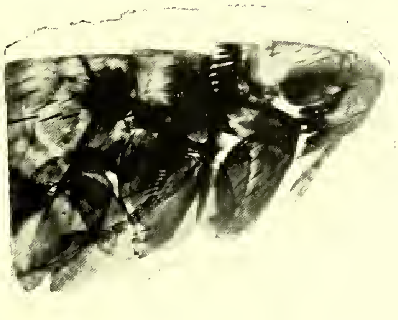
claw × 50

1.—THE HUMAN FLEA (PULEX IRRITANS).



female × 18

male × 18

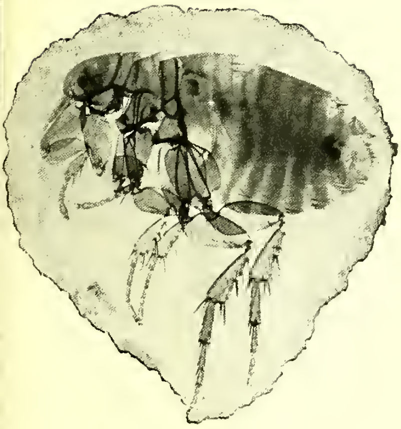


Head of male × 50

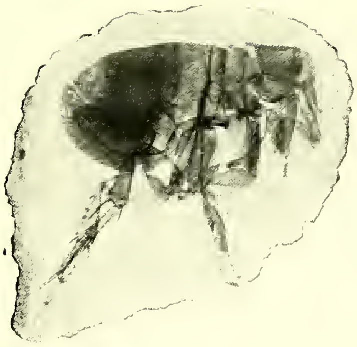
2.—THE BLIND FLEA OF THE RAT (CTENOPSYLLUS MUSCULI).

Dr. Thos. Bancroft.

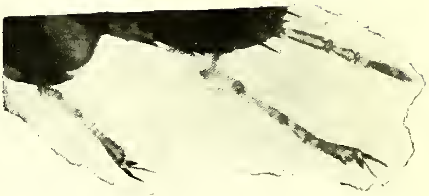
Plate V.



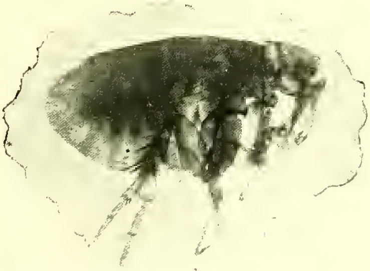
female × 18



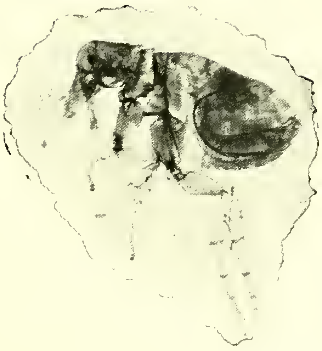
male × 18



claws × 50



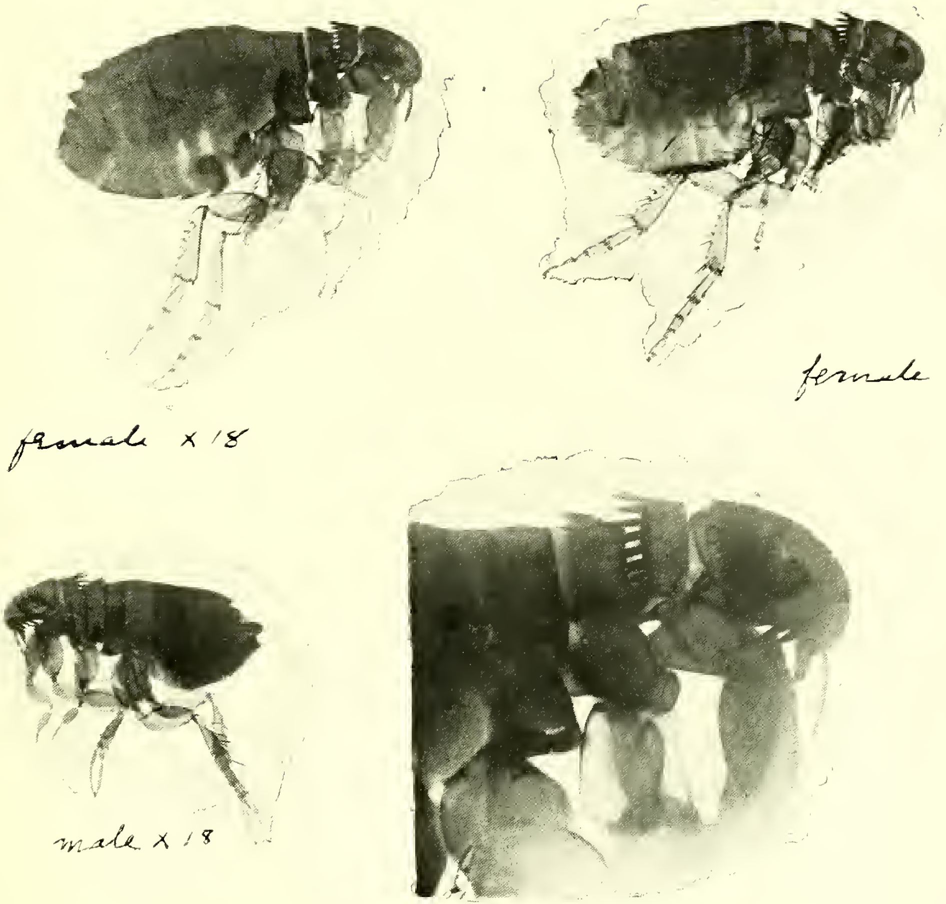
female × 18



male × 18

3.—THE COMMON FLEA OF THE RAT (PULEX CHEOPIS, P. PALLIDUS).

Dr. Thos. Bancroft.



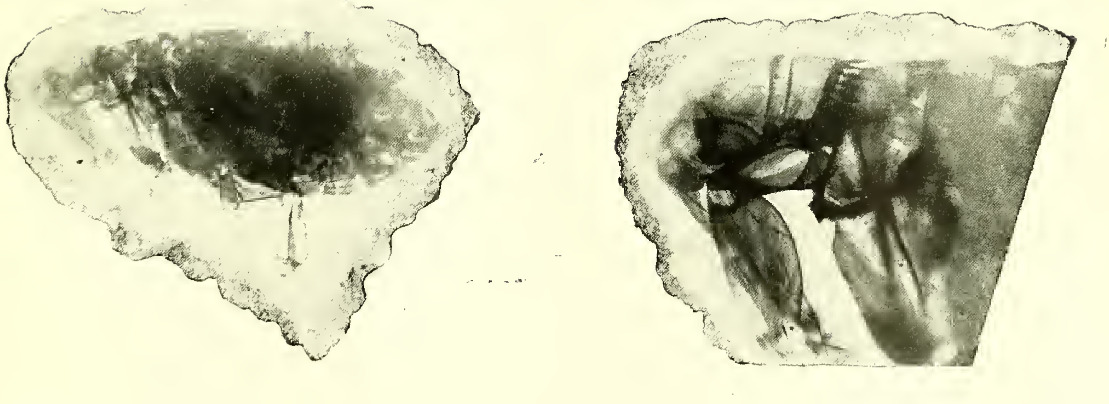
female × 18

female × 18

male × 18

head of female × 50

4.—THE DOG FLEA (CTENOCEPHALUS CANIS, CURTIS).



female × 18

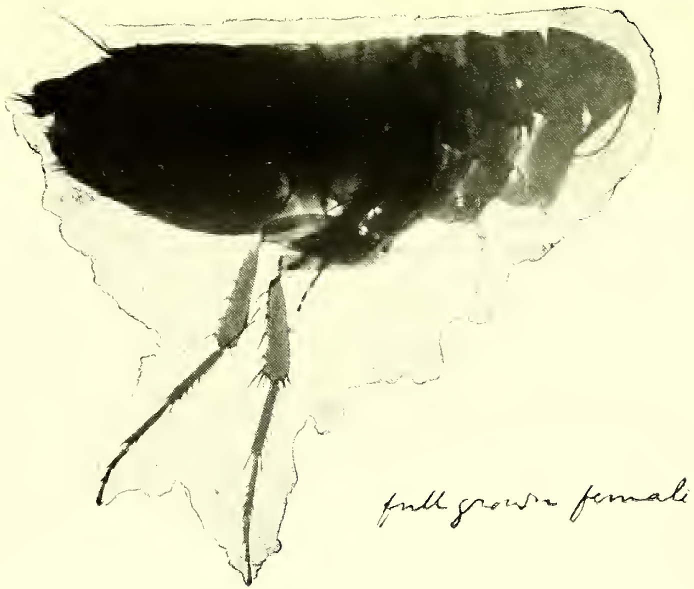
head of female × 50

5.—CERATOPHYLLUS FASCIATUS.

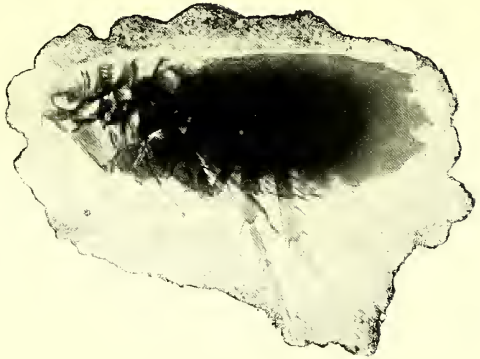
(ONE OF THE RAT FLEAS; VERY RARE IN BRISBANE.)

Dr. Thos. Bancroft.

Plate VII.



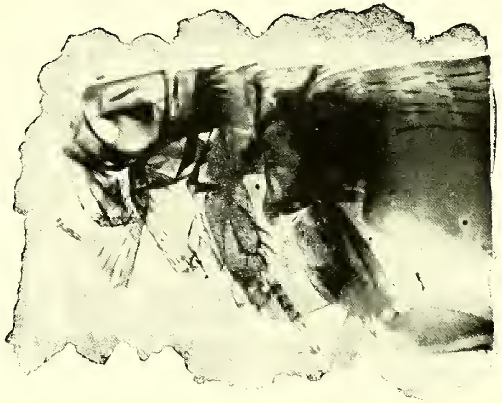
full grown female × 18



young female × 18

male × 18

male × 18



head of male × 50

6.—FLEA OF THE WATER RAT (HYDROMYS. CERATOPHYLLUS SPECIES).

Dr. Thos. Bancroft.

Plate VIII.

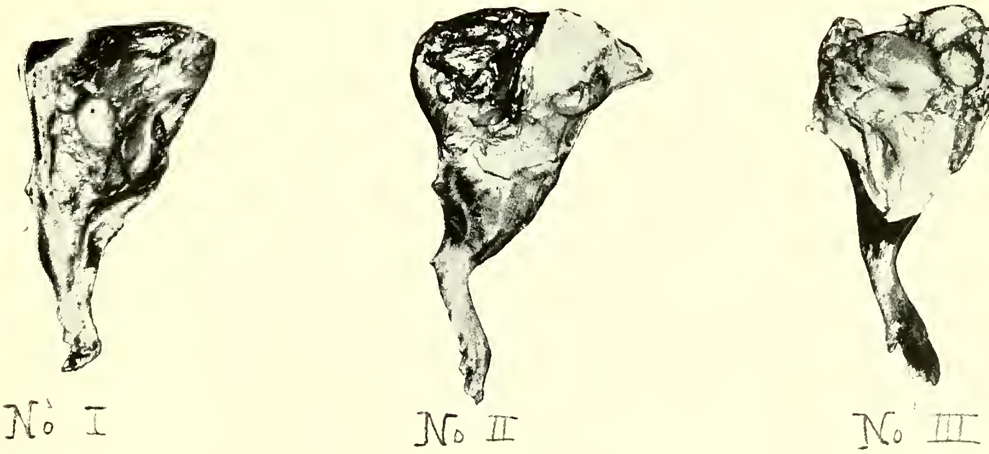


Fig. VI.

Dissections of fore and hind legs of naturally-infected plague rats, showing in No. I. enlarged axillary gland, and in Nos. II. and III. enlarged femoral gland.

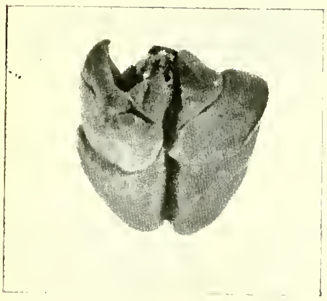


Fig. VII.

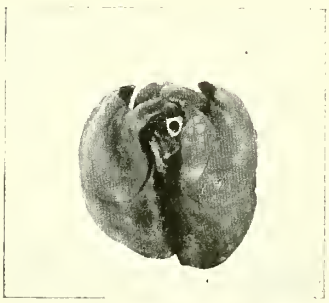
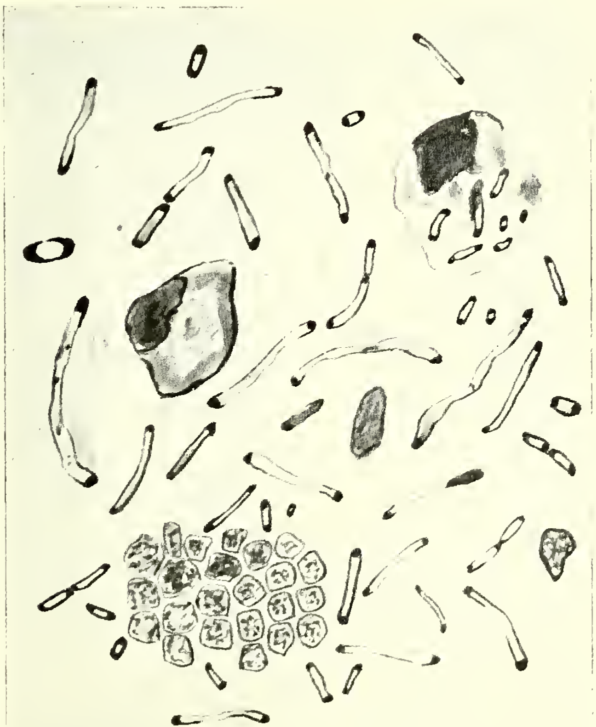


Fig. VIII.

Lungs of two naturally-infected plague rats, showing general congestion and a few scattered grey nodules.



(Diagrammatic, and not drawn to scale.) *C. J. Pound.*

Diagram of contents of stomach of a flea (***Pulex felis ?***). This flea, two days previous to its dissection, had been allowed to suck blood from a plague-infected guinea-pig. The rough sketch shows the abnormally elongated slender ***Bac. pestis***, two large mononucleated cells, one of which has several small plague bacilli in its interior; also, a cluster of cubical cells, which form the lining membrane of the flea's stomach.

Note.—After removal from the plague guinea-pig, this flea was placed upon a healthy guinea-pig, causing the death of the latter from acute plague within four days.

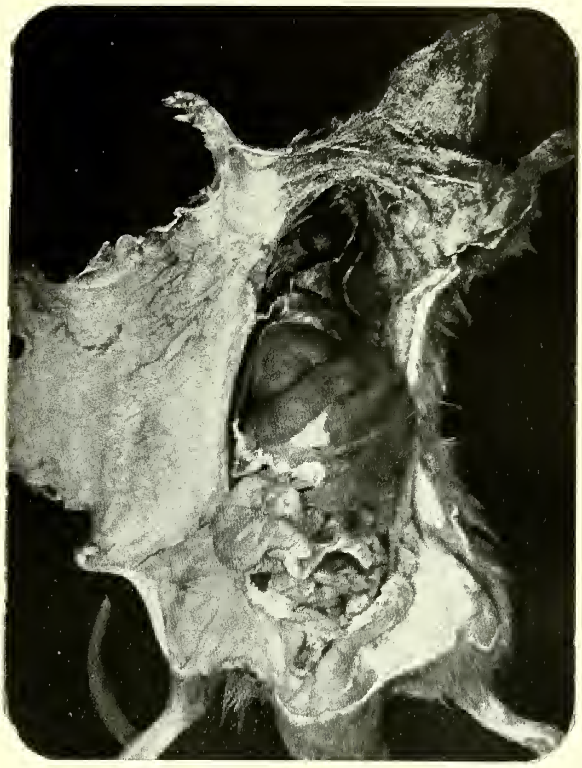


FIG. I.

Naturally-infected rat, dissected to show the principal pathological appearances. Note the enlargement and congestion of the lungs and liver, the enlargement of the spleen, and the axillary and femoral glands; also, the injected blood-vessels of the subcutaneous tissue in the deflected skin of the abdomen.

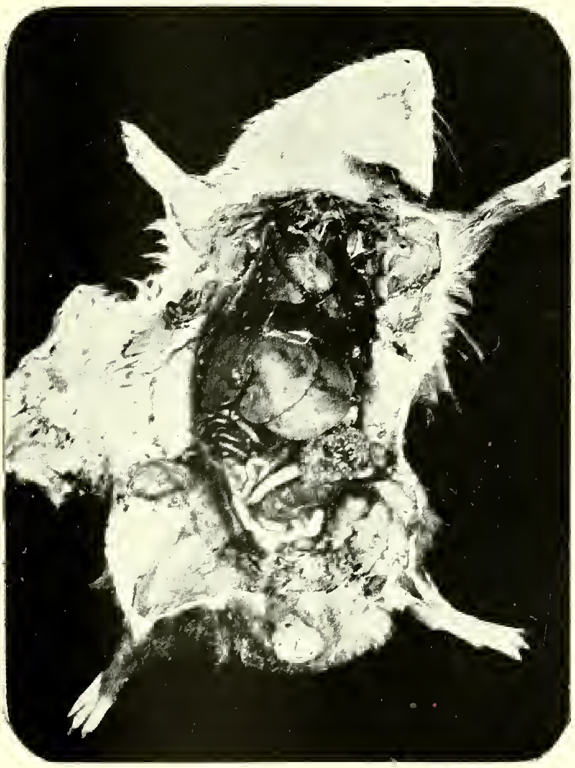


FIG. II. *C. J. Pound.*

Dissection of a guinea-pig (fourth day) inoculated with plague bacilli from a human case. The most conspicuous feature is the necrosed condition of the enlarged friable spleen, which closely resembles miliary tuberculosis. The liver is enlarged, and shows a few necrosed patches. The lungs are congested, with a few scattered grey consolidations. The lymphatic glands situated in the right groin, near the seat of inoculation, are considerably enlarged and hæmorrhagic, while the surrounding tissue shows much hæmorrhagic exudation.

DISSECTIONS OF RAT AND GUINEA-PIG.

PLAGUE FACTORS.

ANNUAL MONTHLY MEAN
FOR A
PERIOD of 8 YEARS
1900-7.

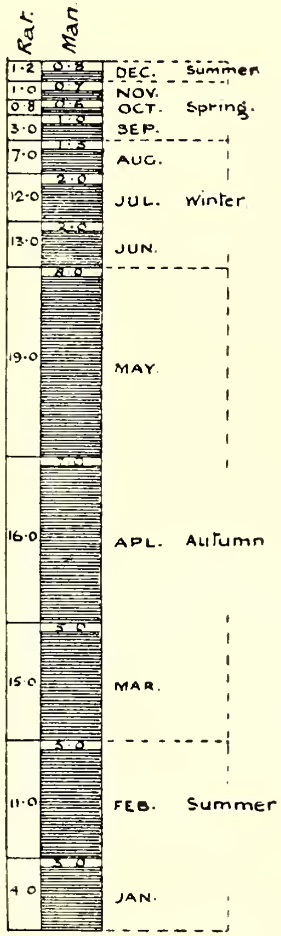
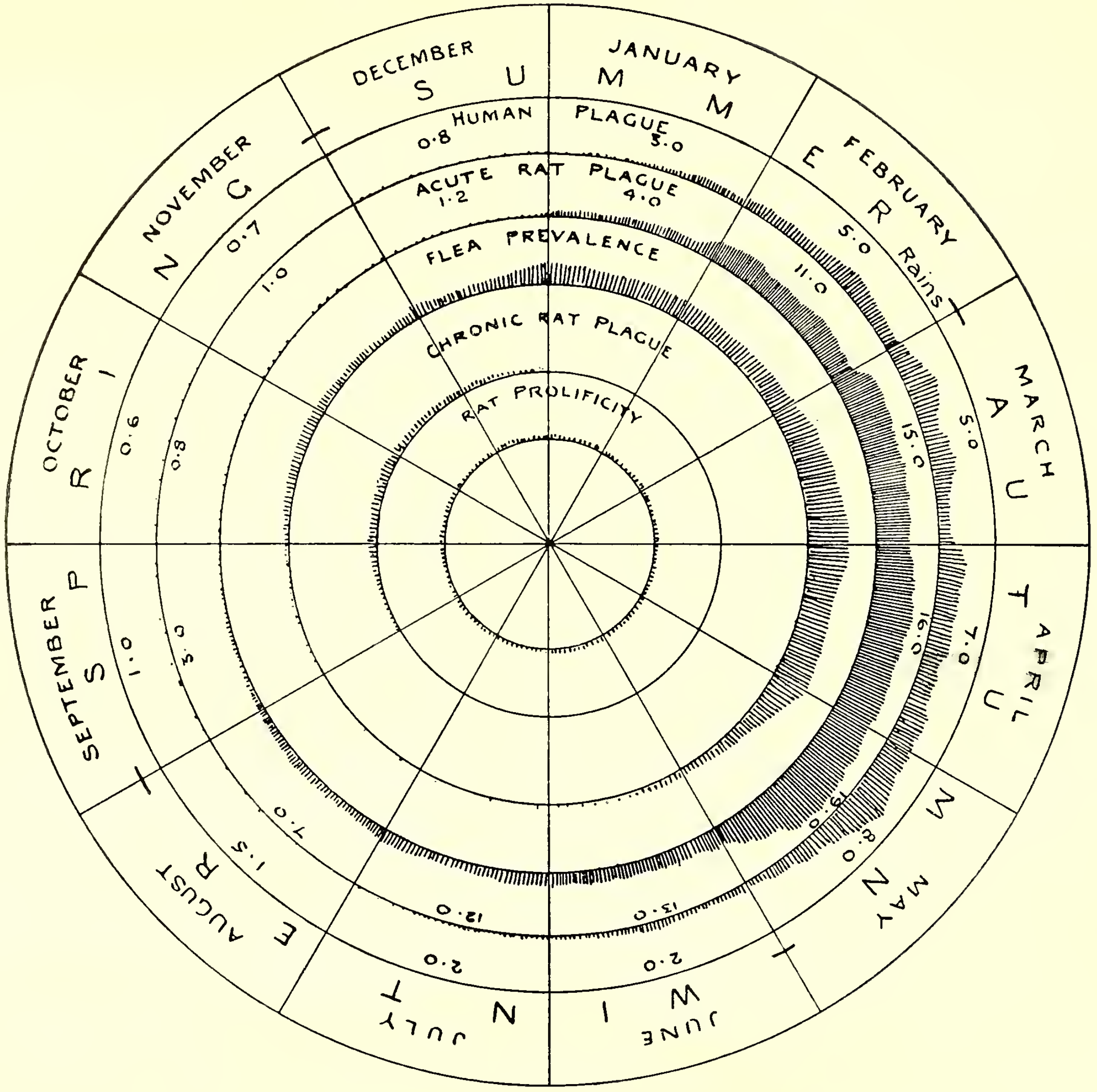


CHART of a PLAGUE-CYCLE in BRISBANE

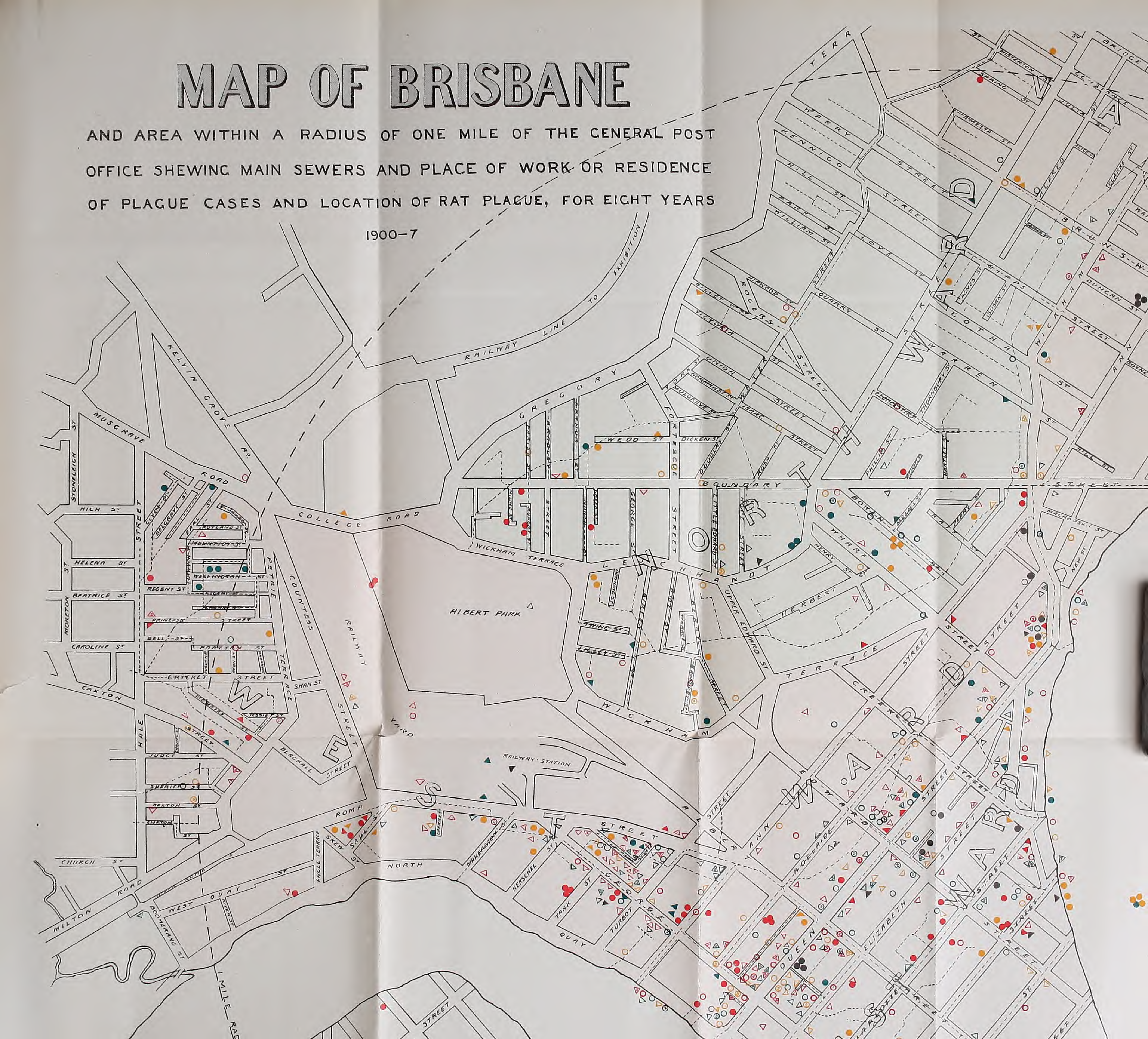
Purely diagramatic, the thickness of the shaded portions only represents approximately, the increase & decrease.



MAP OF BRISBANE

AND AREA WITHIN A RADIUS OF ONE MILE OF THE GENERAL POST OFFICE SHEWING MAIN SEWERS AND PLACE OF WORK OR RESIDENCE OF PLAGUE CASES AND LOCATION OF RAT PLAGUE, FOR EIGHT YEARS

1900–7



HALE
JUDGE ST
SHERIFF ST
SEXTON ST
JESSIE ST
STREET
BLACKALL STREET
YARD
RAILWAY STATION
ROMA STREET
SKEW ST
EAGLE TERRACE
NORTH QUAY
CHURCH ST
MILTON ROAD
WEST QUAY
BOOMERANG ST
HERSCHEL ST
TANK ST
TURBOT
QUAY
GEORGE
WICKHAM
ALBERT STREET
ANN
ADELAIDE
EDWARD
QUEEN
ELIZABETH
CHARLOTTE STREET
MARY
MARGARET
ALICE
CREEK STREET
WILLIAM STREET
SHORT ST
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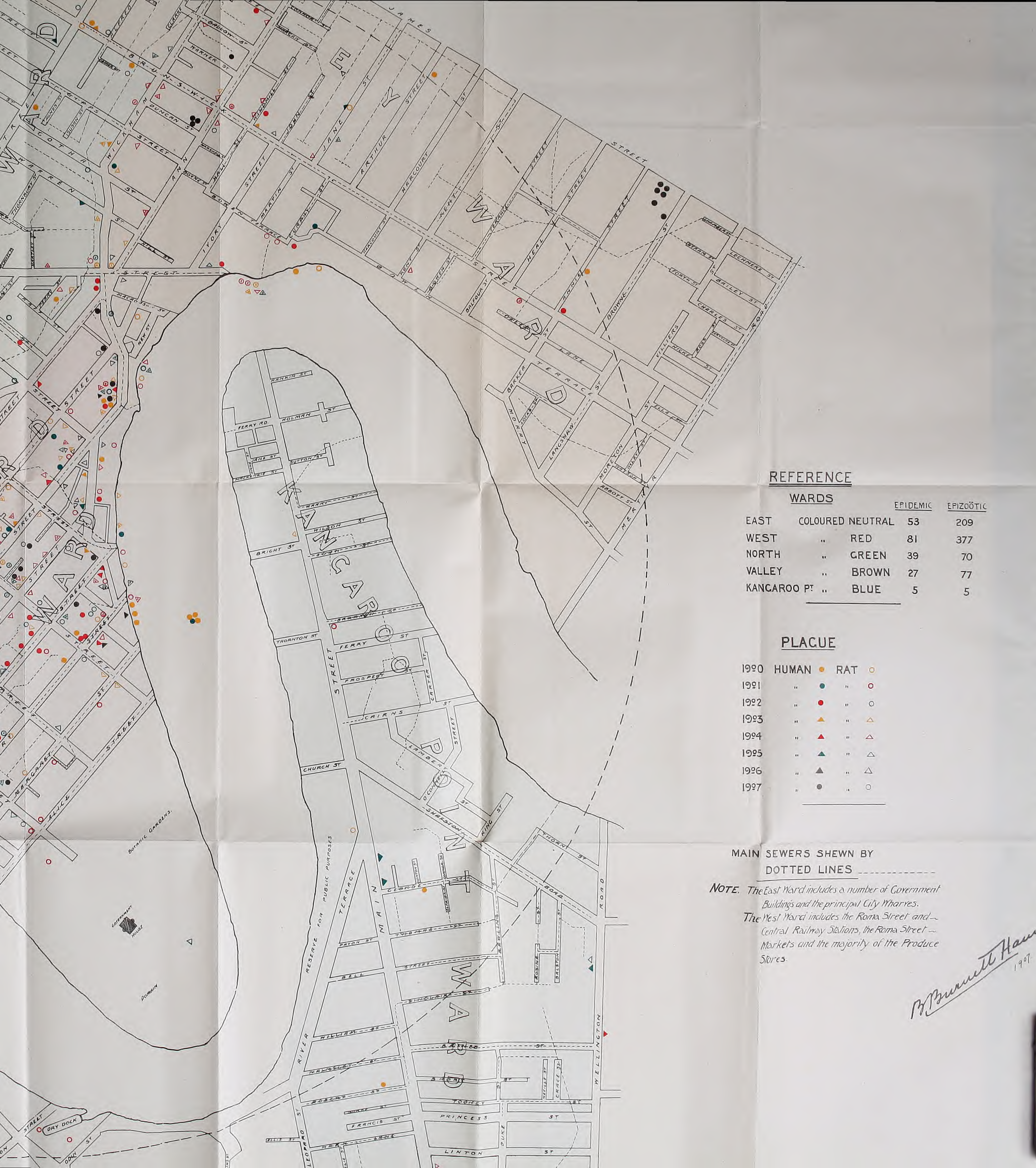
EAST WARD
1 MILE RADIUS
VICTORIA BRIDGE
STANLEY
GREY STREET
HOPE STREET
PEEL
MERIVALE
CORDELIA
MANNING
MONTAGUE STREET
MELBOURNE STREET
MONTAGUE ROAD
BOUNDARY STREET
EDMONDSTONE
STEPHENS
RUSSELL
MUSGRAVE PARK
GLENELG STREET
SOUTH GLENELG STREET
O'CONNELL ST
ERNEST STREET
TRIBUNE
SIDON STREET
DRY DOCK
JANE STREET
HORAN ST
VULTURE
BOTANIC GARDENS
GOVERNMENT HOUSE
DOMAIN



REFERENCE

WARDS		EPIDEMIC	EPIZOÖTIC
EAST	COLOURED NEUTRAL	53	209
WEST	" RED	81	377
NORTH	" GREEN	39	70
VALLEY	" BROWN	27	77
KANGAROO PT	" BLUE	5	5

PLAGUE

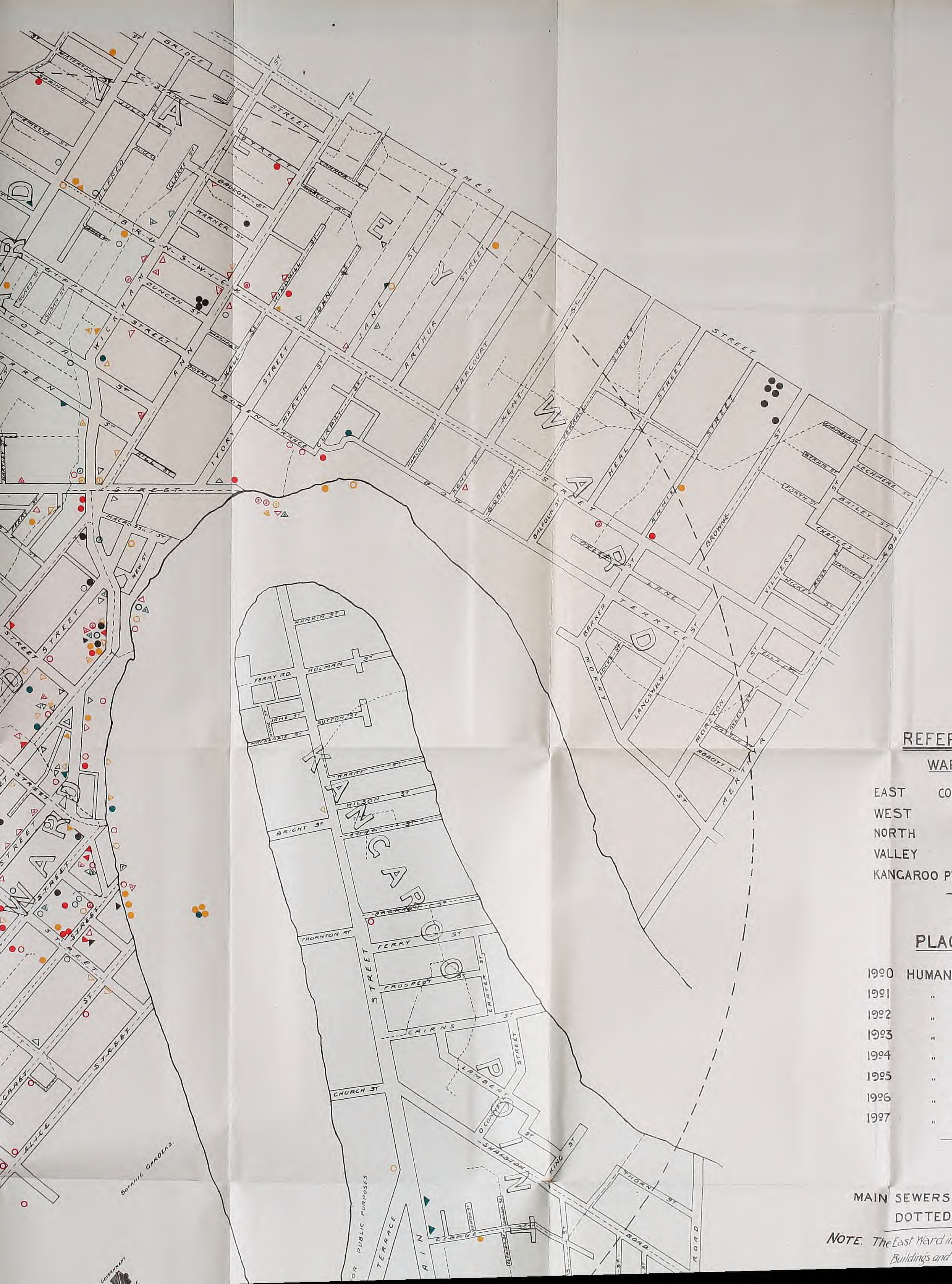
1900	HUMAN	●	RAT	○
1901	"	●	"	○
1902	"	●	"	○
1903	"	▲	"	△
1904	"	▲	"	△
1905	"	▲	"	△
1906	"	▲	"	△
1907	"	●	"	○

MAIN SEWERS SHEWN BY DOTTED LINES ------------

NOTE. The East Ward includes a number of Government Buildings and the principal City Wharves.
The West Ward includes the Roma Street and Central Railway Stations, the Roma Street Markets and the majority of the Produce Stores.

B Burnett Ham
1907

MAP A.



REFERENCE

WARDS			EPIDEMIC	EPIZOÖTIC
EAST	COLOURED	NEUTRAL	53	209
WEST	"	RED	81	377
NORTH	"	GREEN	39	70
VALLEY	"	BROWN	27	77
KANGAROO PT	"	BLUE	5	5

PLAGUE

1900	HUMAN	●	RAT	○
1901	"	●	"	○
1902	"	●	"	○
1903	"	▲	"	△
1904	"	▲	"	△
1905	"	▲	"	△
1906	"	▲	"	△
1907	"	●	"	○

MAIN SEWERS SHEWN BY DOTTED LINES ------------

NOTE. The East Ward includes a number of Government Buildings and the principal City Wharves.

BRISBANE AND SUBURBS

SHEWING LOCATION OF PLAGUE CASES FOR EIGHT YEARS IN METROPOLITAN AREA OUTSIDE ONE MILE RADIUS OF GENERAL POST OFFICE - 1900 - 7.

MAP B.

20 15 10 5 0 20 40 60 Chains

Printed at the Govt. Printing Office Brisbane.

VICTORIA PARK
BOWEN PARK
Brisbane Hospital
Government Domain Reserve
Brisbane Grammar School
Girls Grammar School
ALBERT PARK
PETRIES BIGHT
SHAFSTON REACH
TOWN REACH
HAMILTON REACH
BULIMBA REACH
BULIMBA
HUMBUG REACH
Kinellan
Mowbray Park
NORMAN CREEK
Reserve for Cemetery
Botanical Gardens
Gardens Point
Musgrave Park
Woolloongabba Reserve
SOUTH BRISBANE RAILWAY
Gaol Reserve
School Endowment
Reserve for Orphanage
DUTTON PARK
South Brisbane Cemetery
ST LUCIA REACH
MILTON REACH
TOOWONG REACH
TOOWONG CREEK
TOOWONG
BRISBANE GENERAL CEMETERY
ITHACA CREEK
Red Hill
Ithaca
Normanby
Garfield
Paddington
Milton
Auchenflower
Boomerang
Baroona
Rosalie
Bishopsbourne
Coorparoo Racecourse
Eastleigh
Hackerton
Woodhill
Langlands
Sports Ground
Eden Glen
Tamana
Wahcumba
Hamilton
Elysium
Merthyr Road
SOUTH COAST
RIVER
BRISBANE
SOUTH

—— Reference ——

PLAGUE CASES		WARDS - CITY OF BRISBANE		
1900	●	East	coloured thus	🟨
1901	●	West	" "	🟥
1902	●	North	" "	🟩
1903	▲	Valley	" "	▨
1904	▲	Kangaroo Pt	"	🟦
1905	▲	Merthyr	" "	🟥
1906	▲	Cintra	" "	▨
1907	●			

B Burnett Ham
1907

1900	JANUARY.	FEBRUARY.	MARCH.	APRIL.	MAY.	JUNE.	JULY.	AUGUST.	SEPTEMBER.	OCTOBER.	NOVEMBER.	DECEMBER.
Rats examined	—	—	13	4	1	46	110	109	96	152	120	84
Per Cent infected	—	—	38·46	75·00	—	32·60	19·00	22·02	15·62	4·60	—	—

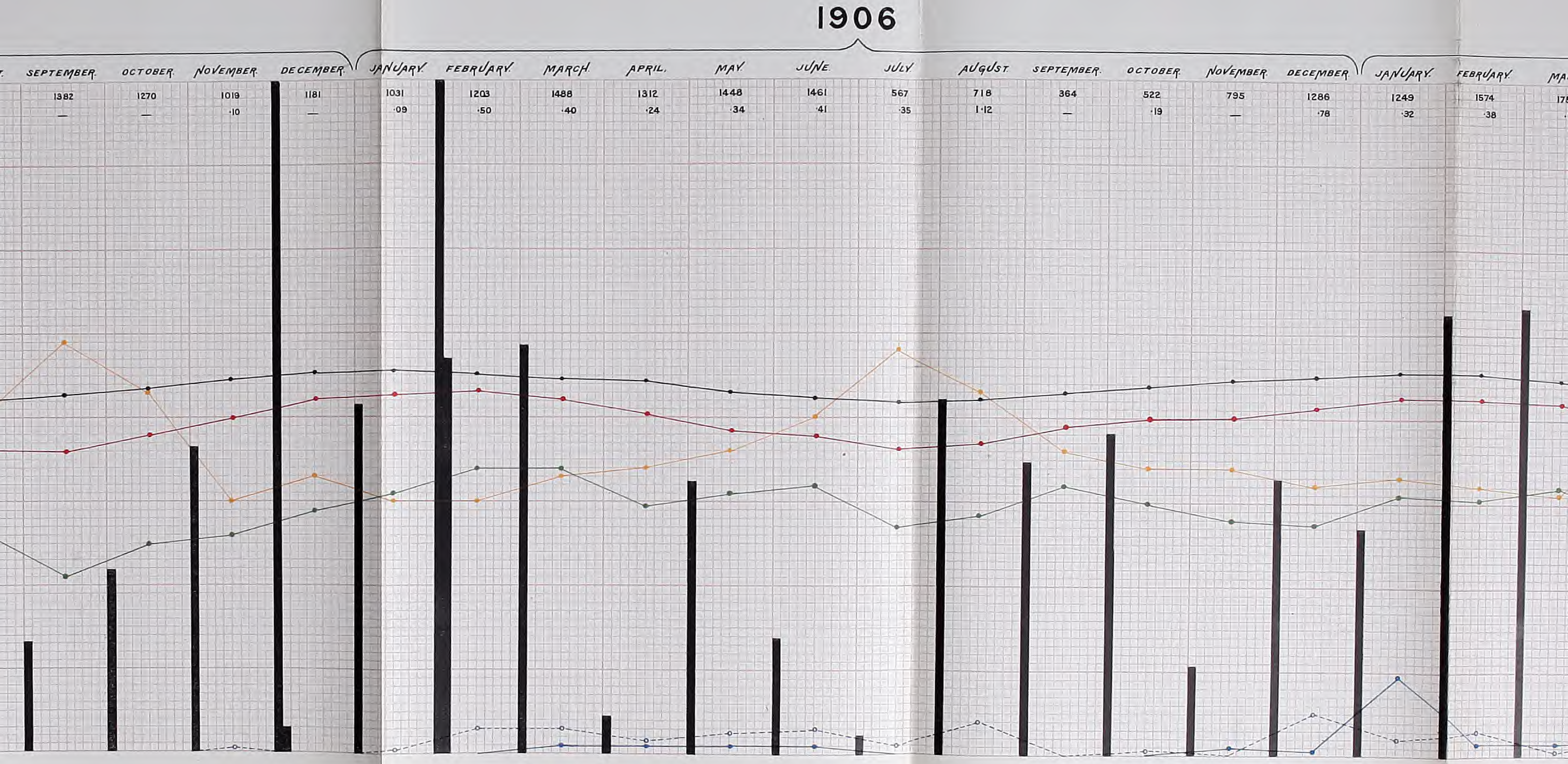
1901	JANUARY.	FEBRUARY.	MARCH.	APRIL.	MAY.	JUNE.	JULY.	AUGUST.	SEPTEMBER.	OCTOBER.	NOVEMBER.	DECEMBER.	JANUARY.	FEBRUARY.	MARCH.
Rats examined	44	—	48	169	163	121	144	136	245	332	273	176	236	246	156
Per Cent infected	—	—	—	9·47	14·10	26·45	13·89	5·88	·40	—	·37	—	1·27	2·44	4·49

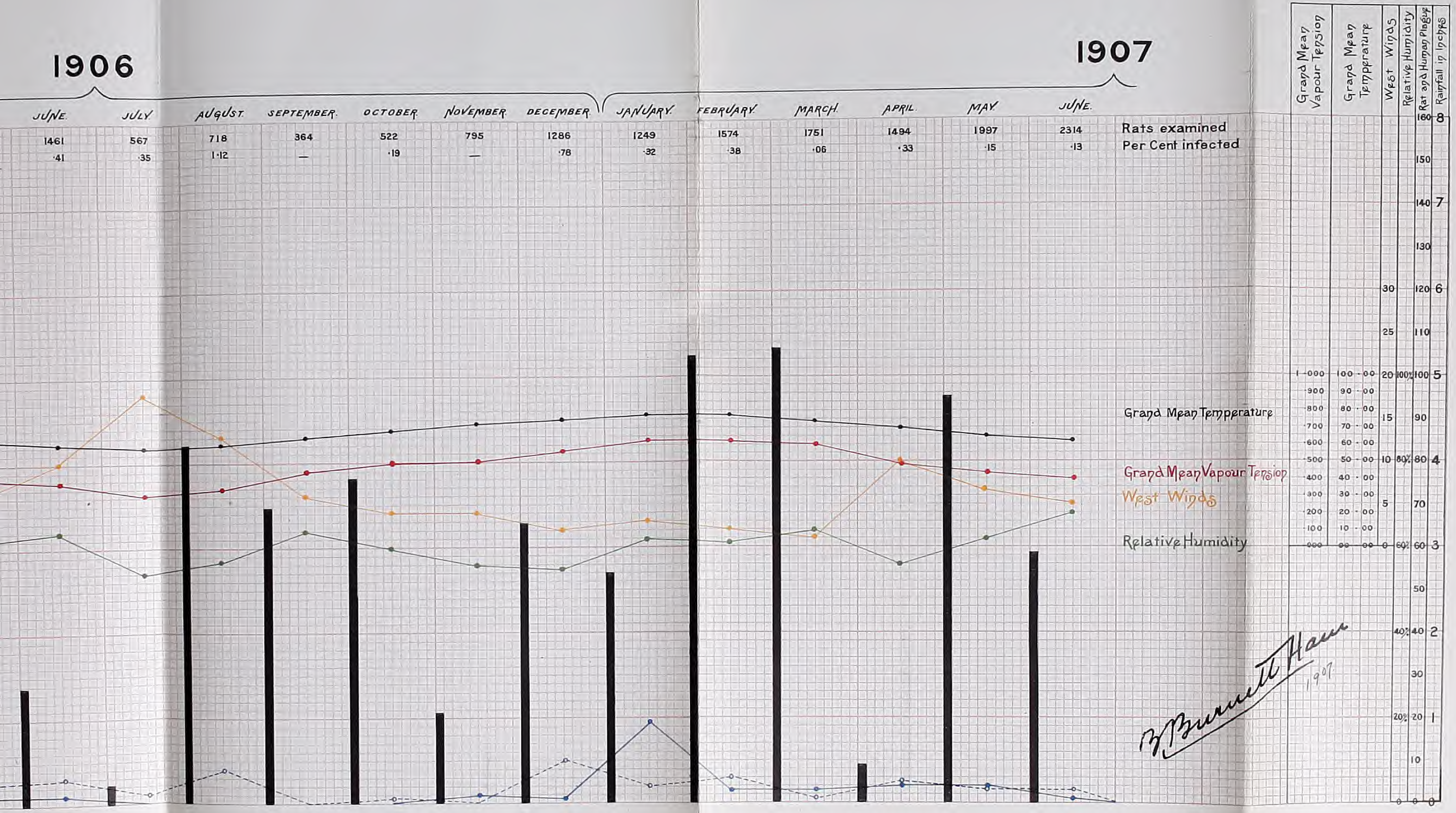
SEASONAL PREVALENCE OF PLAGUE,

Rainfall ▮, Plague in Humans —, Plague in Rats ----, Grand Mean Temperature

OF PLAGUE, BRISBANE, METROPOLITAN AREA.

Grand Mean Temperature —, Grand Mean Vapour Tension —, Relative Humidity —, West Winds —,


1906
SEPTEMBER.
1382
—
OCTOBER.
1270
—
NOVEMBER.
1019
·10
DECEMBER.
1181
—
JANUARY.
1031
·09
FEBRUARY.
1203
·50
MARCH.
1488
·40
APRIL.
1312
·24
MAY.
1448
·34
JUNE.
1461
·41
JULY.
567
·35
AUGUST.
718
1·12
SEPTEMBER.
364
—
OCTOBER.
522
·19
NOVEMBER.
795
—
DECEMBER.
1286
·78
JANUARY.
1249
·32
FEBRUARY.
1574
·38



B Burnett Ham
1907